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### THE RÔLE OF THE PSYCHIATRIC DISPENSARY: A REVIEW OF THE FIRST YEAR'S WORK OF THE DISPENSARY OF THE PHIPPS PSYCHIATRIC CLINIC.\*

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The problems of the dispensary differ somewhat from those of the clinic and the aim of this review is to discuss some of these problems in the light of the first year's experience in the dispensary of the Phipps Psychiatric Clinic. It is obvious that in the dispensary it is not possible to carry out the same detailed studies which are possible when the patient is under continuous observation in the hospital; the examination is frequently somewhat summary and conclusions have to be drawn from data which are recognized to be inadequate for the thorough understanding of the case. The limitation of time tells especially in psychiatric work, for in this department we cannot always plunge abruptly into an examination; to establish satisfactory relations with the patient is essential for good results, but this requires time; the speed of the examination is largely dependent upon the condition of the patient; the physician is occasionally not permitted by the patient to take notes of important facts. The fact that the anamnesis, so important in psychiatric work, is often given by some friend with little knowledge of the case, helps to make the interpretation of many cases still more uncertain. It is not, therefore, from the dispensary that one expects a psychiatric discussion based on well-analysed clinical material. In the dispensary the

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practical demands of the patient stand in the foreground, and these demands usually require the investigation of a whole situation and not merely that of the patient as a unit; the attempt to modify this situation, which is essential for the satisfactory treatment of the case, takes one from the dispensary into the home and brings one face to face with the vital problems of the mental hygiene of the community.

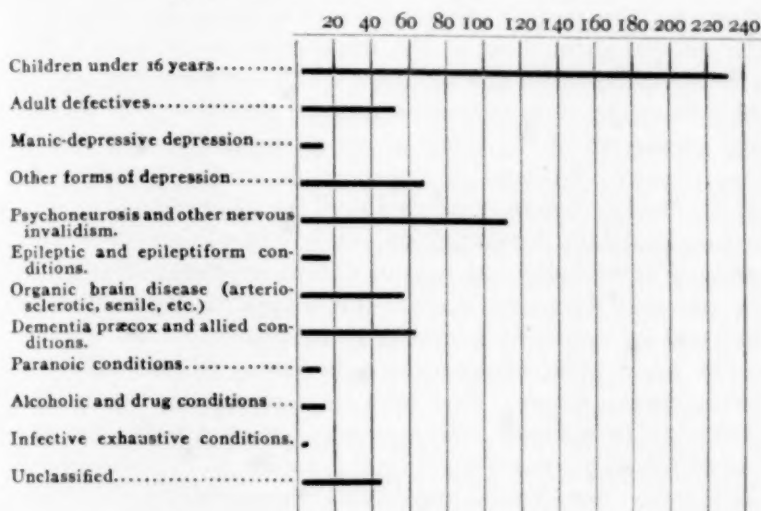
If the inability to make a detailed study of the more complicated cases is at times to be regretted, there is compensation in the treatment of many interesting minor forms of maladjustment, which are included in the stream of dispensary material. The fact that so many of the cases present disorders to which it is hard to give a satisfactory name, and to group in any of the conventional diagnostic groups, is perhaps partly to be understood in the light of the changing conditions of psychiatric work. The descriptions of mental disorders found in classical text-books have been chiefly based on the clinical material found in the hospitals for the insane, while the less severe but more common forms of maladjustment have received scant attention, and are even somewhat grudgingly allowed to belong to this special branch of medicine. It is therefore not surprising that there is little agreement at present as to the formulation of the diagnosis in many such cases. In the work of the dispensary the aim has been rather to formulate the diagnosis of the cases in intelligible working terms rather than in terms of some rigid classification which does violence to the facts. It will be some time before we are in a position to diagnose our cases in uniform terms which will do justice to the dynamic factors in the individual case and at the same time indicate the relationship of the case to recognized forms of disorder.

During the past year (May 1, 1913, to April 30, 1914) 708 patients registered at the dispensary; in the accompanying chart the distribution of the patients in large provisional groups is shown.

By far the largest group is that of children under 16 years of age; it contains 236 patients. No more important nor more fundamental problems are brought before the dispensary than those furnished by this group of cases, which even numerically is so striking; it raises important questions as to the relation of the functions of the dispensary to the work of other social agencies.



CHART I.—PHIPPS PSYCHIATRIC DISPENSARY. MAY 1, 1913, TO APRIL 30, 1914. CLASSIFICATION OF PATIENTS (708).



In close connection with the problems of these children may be discussed the group of adults in whom the crucial factors are mental defects similar to those which bring a large number of children of school age to the dispensary.

Next in numerical importance to the group of the children is a large group of cases including not only well-recognized types of psychoneurosis, such as hysteria and obsessional states, but also a great variety of cases of nervous invalidism; in these cases of invalidism the mechanisms of the psychoneuroses are also to be traced, but the clinical picture is less distinct and is largely determined by special constitutional traits of the individual.

In another group is brought together a great variety of cases, where a reduction of mood or definite depression is in the center of the clinical picture; many of these cases might no doubt with equal propriety be brought into relation with the above group of cases of nervous invalidism. From this group of depressions it is possible to separate another group of 13 cases, which present in sufficiently clear form the manic-depressive type of depression (these, along with one case of excitement and two cases of the mixed type, form the total of 16 cases of manic-depressive in-

sanity). To give this familiar name to these cases does not necessarily mean that we know more about them than we do about the other group of depressions; in fact, it is apt to paralyze our thought about them, as the familiar diagnostic term brings with it an assumption of knowledge which is rather illusory. To think therefore of these manic-depressive depressions in the general framework of the whole group of depressions will help to keep alive the problems of the manic-depressive disorders; instead of trying to grasp the meaning of the other cases in relation to the superficially familiar group, we shall be encouraged to work seriously at the individual cases of the larger group and to utilize the principles discovered there to throw light on the familiar but little-understood manic-depressive depression.

The larger group of depressions consists of 68 cases of rather heterogeneous nature. One must here repeat that these cases have been studied under dispensary conditions and that this presentation merely gives a provisional grouping of these cases; this whole group furnishes an exceedingly fruitful field for intensive work and there is no doubt that in a large number of these cases a fairly satisfactory reconstruction of the whole process could be arrived at. Without such an analysis we are working with rather crude measures.

A provisional division of this group was made into two subordinate groups; the former of these was characterized by the prominence of somatic complaints, such as headache, dizziness, crawling feeling, vague pains and paræsthesias, "nervous indigestion," constipation and sleeplessness.

In the other sub-group were included all cases of depression not otherwise classified. With regard to these two subordinate groups of depression, it is probably of significance to note that in the former group, with the marked prominence of somatic symptoms, only 12 out of the 37 patients were under 40 years of age, while in the second group 19 out of 31 were under 40. With regard to the sex-incidence, in the former group 28 out of 37 patients were women; in the latter group 19 out of 31 were women.

The symptom-picture, if one is entitled to use this term for symptoms so poorly knit together, in the first group is thus seen to be more frequently found in women and at a somewhat more advanced age than the symptom-pictures of the second group. To

many of these cases it is evident that the term "involution-melancholia" may be applied without doing too much violence to our conscience; but the comfort supplied by this familiar term does not bring with it much illumination and again is apt to dull the interest in research. As a matter of fact, none of the patients presented the classical picture of involution-melancholia, and it would have required a certain classificatory enthusiasm to have grouped many of the cases under this head. When one turns from these more formal points to the consideration of the individual cases, we find it possible, even under dispensary conditions, to get considerable insight into the development of the symptoms and into the rôle which the symptoms play in the adjustment of the individual to the actual problems of life. Here the study of the personality and of the actual situation is essential; it is usually necessary to go beneath the compromise of the individual life to study the actual dynamic factors. It was striking to see in how many cases the symptoms seemed to arise in the setting of a difficult marital situation.

The line of treatment follows the above general indications, and the difficulties in the way of a satisfactory result are obvious. The personality of the patient is not easy to modify at this period; faulty adjustments have become habitual; compromises and evasions have become an integral part of the individual's life; the symptoms have to a certain extent proved to be along the line of least resistance; the marital situation has frequently to be accepted, even although we find that the marriage was originally entered into without affection in order to forget a first love. Frequently, however, a certain modification is possible; the break in compensation may have been due to factors which can be modified; the physical health of the patient can perhaps be improved, faulty hygiene corrected, sane recreation and wider interests introduced into a rather dreary and narrow life, and the patient brought to face in a more direct manner the underlying factors. By means of the social service department it is often possible to improve the situation at home, and some tactful advice to the husband or wife or family of the patient may bring good results; an occasional visit to the park or even to moving pictures is a prescription not beneath the dignity of the physician and may be based upon more rational grounds than a routine tonic or hypnotic.

In the more heterogeneous second subordinate group of depressions the same principles apply as in the group already discussed. Here, too, the more opportunity one has of studying the individual psychology of the case the further does one get from the standpoint of formal classification and the more anxious one is to formulate the disorder in dynamic terms. To illustrate what has been said, the following case may be briefly reported:

J. S., a man 68 years of age, had not been very successful financially for some time. He came to the dispensary because he did not rest well; he had swimming of the head, shortness of breath and some nausea. He himself referred the onset of the symptoms to financial troubles which began in 1911. He felt sad, but there was no retardation. He was restless during the examination. His memory was very good. Physically, there was little to notice; he showed very slight arteriosclerosis.

The case appeared to be a fairly simple type of depression, partly explained by his financial worry, and more or less intelligible as an excessive reaction to this worry in a person of advanced age.

In this case the opportunity for a fuller review of the patient's life brought up very important factors, which would have to be taken into consideration before one had a right to formulate the exact nature and mechanism of the disorder.

It was found that the patient had had earlier in life several attacks of depression. These attacks of depression, however, were not merely to be looked upon as the expression of some obscure constitutional disorder. They were found to be directly related to the problems of his sexual life. The patient had worried considerably about masturbation. His physical health was running down, and at the age of 20 he went to a quack physician, who diagnosed some disease of the bladder and of the nervous system, whereupon the patient went to bed for two months. He had several similar weak spells during the following years. He married at the age of 26, and from that period until the age of 66 (in 1911) he was absolutely free from depression.

The case illustrates the limitations of dispensary work and the wisdom of making the diagnosis in rather simple and common-sense terms. If the examination had been confined to the first two interviews the case might have been considered a reactive depression, to be explained partly by the organic changes so frequent

at this period of life. The later discovery of the earlier attacks might have seemed to justify the designation of manic-depressive depression. The understanding of this case could only be attained by the analysis of the individual attacks, and in the light of the mechanism of the earlier attacks the onset of the depression at 66 became a problem of much greater interest than one of classification.

The largest group of adult patients, as already mentioned, consisted of those who either presented a well-recognized psychoneurosis or less well-defined forms of nervous invalidism; the group is obviously very far from homogeneous. Several cases could perhaps with equal propriety have been included in the group of the depressions, especially those showing a prominent hypochondriacal trend, and several cases grouped under the depressions might no doubt on a fuller examination have been found to belong to the psychoneurotic group. The problems of the psychoneuroses are sufficiently difficult even under the most favorable conditions of study and treatment; the diversity of opinion with regard to their fundamental mechanisms is extreme and the nomenclature is in a rather fluid state.

The cases observed during the year fell into six provisional groups as seen in the following table:

PSYCHONEUROSES (INCLUDING ILL-DEFINED TYPES OF NERVOUS INVALIDISM) .....	110
Anxiety-neurosis .....	21
Hysteria with attacks or purely physical symptoms.....	23
Hysteria with morbid fears.....	19
Obsessive thoughts and actions.....	14
Hypochondriacal and neurasthenic states.....	13
Nervous invalidism of less well-defined type (frequently inadequate data) .....	20

It would be out of place here to take up the more fundamental problems of the psychoneuroses; it may be useful to discuss how far the conditions of dispensary work introduce special complications. These cases will always be an important factor in dispensary work, because, owing to the less severe nature of their symptoms, the patients frequently are disinclined to come into a hospital, and because their treatment is as a rule too prolonged for continuous hospital residence.

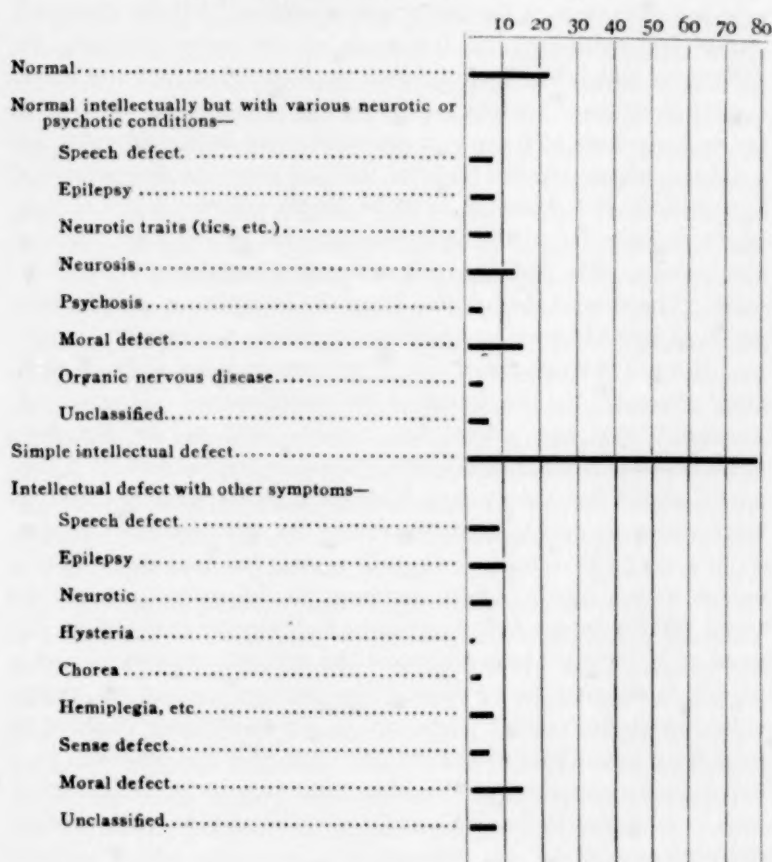


Whatever may be one's views as to the nature of the psychoneuroses, most will agree on the necessity of a rather thorough review of the life of the patient in all its bearings. The special constitutional traits of the patient, the details of the individual life, have to be understood and the patient has to learn to bring to discussion much that has hitherto been strenuously repressed. There is no short-cut towards the necessary self-knowledge and any attempt to force the pace or to change the tedious step-by-step progress is apt to retard rather than accelerate the recovery of the patient. Re-education can never be made an abrupt process and the attempt to do so is futile. As the physician cannot delegate the work to any other person, but has to continue the detailed study and treatment of the case personally, great demands are made on his time. The possibility of treating the dispensary patients along these lines has been due to the comparatively large number of physicians who are associated in the work of the dispensary. It not infrequently happens, however, that for various reasons rather strict limitations of time are put on the treatment of the patient; the patient may perhaps be spending a short period in Baltimore, or the demands of the other patients may not allow the physician to give the desired time to the special patient. There is a tendency in some quarters to dogmatize about the minimum time necessary for satisfactory treatment of these patients and the impression might be gained that, if these time conditions were not fulfilled, the treatment of the patient would be rather futile. There is of course much truth in this caution against expecting quick results. On the other hand, the physician must not demand too much. It is quite striking what excellent results may be gained from a rather limited series of interviews. The record of the case will of course not be a very convincing clinical document, and many interpretations may be borrowed from the physician's experience with other cases; the therapeutic result, however, may be very satisfactory and the patient may return to the home relieved of symptoms, and having acquired a degree of self-knowledge which is the best guarantee of satisfactory mental hygiene in the future. Useful statistical data as to the comparative results of the different forms of treatment of the psychoneuroses are not available; results rapidly attained may be only transitory, but the impression from the dispensary work has been

that dispensary conditions are not unfavorable for the treatment of this class of patients, and that even under great restrictions of time the physician, in the study and treatment of these disorders, should feel encouraged to make use of the same principles, the validity of which has been demonstrated on other material where a more exhaustive analysis of the case is possible. It may not be out of place here to refer to a practical point of considerable importance; where detailed study of the case over a prolonged period is necessary, the physician must be on his guard against the patient's making use of the interviews for his or her own purpose, and making little real progress towards a satisfactory readjustment. The patient may derive from the interview a gratification, which simply becomes an additional factor in her complex neurosis. It must be emphasized that this situation is not to be immediately attributed to the trend of the examination, for one finds frequently that the patient has already indulged in the same attitude towards other physicians, who have treated her for a prolonged period for her neurotic symptoms along somatic lines. It is possible, no doubt, in many cases, for the physician to deal directly with this situation where it arises; but that depends to a certain extent on the whole make-up of the patient. Here the social service department has been of invaluable assistance. Instead of laying too much weight on the detailed analysis of such a case, it is essential to emphasize the positive side of the reconstruction of the patient's life and to do everything possible to raise the general level of the patient's interests and activities by a thorough investigation of the home situation and practical guidance with regard to definite activities. The social-service worker can here co-operate with the physician in a way which obviates certain very practical difficulties in the relation of the patient to the physician.

Of the total work done in the dispensary, that in relation to children is socially perhaps the most important at present, and promises to develop extensively in the future. The children who came to the dispensary formed exactly one-third of the total number of cases. The conditions for which the children were brought to the dispensary varied very widely, some cases being brought for small habits like thumb-sucking, or for peculiar tics, while others presented conditions of profound defect. These cases have been grouped as is shown in chart 2.

CHART 2.—PHIPPS PSYCHIATRIC DISPENSARY. MAY 1, 1913, TO APRIL 30, 1914. CHILDREN UNDER 16 YEARS (236).



Twenty-five children were found to be practically normal. Some of these were examined because they were members of families where many cases of defect had been found; others were brought by teachers on account of difficulties at school; others were brought for a thorough examination before the child was placed in some home. The children who presented distinct anomalies (211 in number) have been grouped according as they did or did not show a definite intelligence defect, this usually being standardized by means of the Binet-Simon scale. One hundred

and forty-eight children showed a definite intelligence defect, and Dr. Hall has reviewed the cases in this group, and discussed the important problems that they present both from the medical and social point of view.

The children who did not show a definite intelligence defect presented a great variety of clinical problems.

The importance of the early period of development in relation to the origin of nervous and mental disorders in the adult period has steadily gained recognition as the study of the adult psychoses has become more thorough. In studying the individual adult psychosis there may always be some uncertainty as to how far the memories of the actual experiences of childhood have been distorted in the adult mind. In the group of children at present under consideration one has the opportunity of studying these factors more directly, and one can trace at an early stage the development of faulty habits fostered by the bad hygiene of the environment. Traits, which later on may be considered as inborn and constitutional, are seen in their relation to the actual situation which develops them. The study of these children promises to give us very important information with regard to the development of the child and the evolution of neurotic traits and other anomalies of adjustment.

Several children presented already a well-marked psychoneurosis.

At this early stage the mechanism of the psychoneurosis may be traced much more easily than when it has passed through the later adult elaborations.

J. F., a boy of 7, was referred to the dispensary from the Harriet Lane Home. He had been brought to the hospital, as he had shown peculiar nervous symptoms. In January, 1914, he had suffered from chicken-pox. Four days after recovery he cried out that his feet were growing larger; that his hands, and nose and mouth were growing larger. He began to dream of snakes. On account of this he was afraid to go alone into a room. One day his mother hurried to him, as she heard him crying. He was shaking and crying out that he had seen a black snake coming after him. He was now sensitive and did not want people to look at him. He dreamed frequently of robbers, and in the day-time he was afraid of robbers.

In the second interview at the dispensary the following facts were elicited, the boy talking very frankly, even in the presence of his mother and several physicians:

In his dreams he saw robbers, and robbers would kill his younger brother Bob and cut him up in pieces. He also had dreams that his brother was drowned. The mother said that when the younger brother was born the patient would object to her nursing him, and would try to push the baby away with his feet. The mother stated that the patient was very fond of his brother Bob. The patient, however, made the interesting statement, "Bob is the pet and sits on mother's lap, and if anything wrong happens, I do it." He also said: "Bob is the pet, because he always sleeps next to mother."

In the dispensary the patient made the statement that he had been growing small, just as small as Bob (the brother). Evidently some of the boy's morbid fears were closely related to the repressed jealousy of his brother.

The date of onset of the symptoms was important. It came on after the attack of chicken-pox. Before the chicken-pox he and his brother had shared the mother's bed, Bob sleeping next to the mother. When the patient had chicken-pox he was left alone in the bed, while the mother and Bob slept on a couch. The patient now cannot bear to hear chicken-pox mentioned.

This meager analysis of the case was sufficient to indicate that the boy was to a certain extent suffering from his jealousy and its repression, and the mother was advised to take quite seriously the utterances of the patient, and get him to feel that he could talk to her absolutely frankly about all that was in his mind. It was hoped that the development of the frankest possible atmosphere in the home would prevent the further development of neurotic symptoms.

In another case the clinical picture was a little more ominous, and the family atmosphere very difficult to modify.

The patient, a girl of 8, had developed slowly in infancy, but showed no mental defect. For many months she had suffered from a choreiform condition of rather peculiar nature. She had a strident and unnatural laugh; she asked questions incessantly, and the questions were frequently rather aimless and with self-evident answers, *e. g.*, "Is mother sitting there?" (Looking at her mother.) "Are you my doctor?" "Am I your patient?"

The child showed an abnormal interest in water. On one occasion she had been rescued in the middle of an attempt to go down the sewer. Two months later she pointed to water flowing down the sewer and said: "Water can go down there, but little girls cannot." The flushing of the toilet fascinated her, and her mother had difficulty in keeping her away from the faucets.

This obsessive curiosity of the child was interesting in relation to the strongly repressive atmosphere in which she was brought up, which tabooed many topics of childish interest. The father was strictly religious, unable



to ride on the trolley on Sunday, looking upon dancing as destructive of the soul. The mother was rigidly conventional, and felt that she should punish her two-year-old boy for making reference to his sister being in the toilet. When the patient at home would roll on the floor in a careless way, the mother was horrified at her scandalous behavior.

In this case it was felt that every effort must be made to give a little more freedom to the development of the natural instincts of the child, and the social-service worker has spent much time in trying to interest the mother in studying and trying to understand the child. She is trying to get the mother to give the child freer opportunity for development. The child has made weekly visits to the dispensary, where she plays contentedly with the toys provided for her, and has some respite from the continual nagging of her mother. It may be possible to arrange for the child to have a short holiday away from her parents, although in their anxious care for her they are rather averse to this.

Actual studies of cases of this description will be an important contribution to our knowledge of psychopathology. Such work will have to be done as a rule in the dispensary, for the child will have to be observed for a very long period. Parents are frequently unwilling to be separated from their child, and treatment consists in the modification of the home, as well as in giving direct assistance to the child.

The development of minor neurotic symptoms in relation to faulty environment was well seen in a little girl of 6, who was brought to the dispensary, as she was subject to night terrors. She would wake up screaming "Mamma" (grandmother), and frequently had to be taken into her grandmother's bed. The parents of the patient were dead. With regard to her other symptoms, the patient was said to have comparatively little appetite, and to be somewhat capricious with regard to food. She was also timid if left alone, and would always have to reassure herself as to the whereabouts of her grandmother. As a matter of fact, the grandmother had been abnormally timid as to the child being allowed to go out; she was always afraid that something would happen to the patient. She gave the patient numerous pennies, allowed her to buy candy constantly and to eat indiscriminately between meals. The grandfather thought it was a shame to try and force any food on the patient at meals, and the patient therefore got whatever she wanted; she was always able to get what

she wanted by harping on it, or by crying until the end was obtained. The patient had been given some sleeping medicine by another physician, and the grandmother rather favored the continuance of this. Here the whole education of the patient was seriously at fault. The main problem was to organize the hygiene of the home, and, if possible, to instruct the grandmother, while the child herself required no special treatment.

An extremely important group of cases consists of children who have shown evidence of what may roughly be called moral defect; the patients have either been pilfering, or shown intolerable behavior of one type or another, or have shown sexual immorality of a more serious nature than mere incidental masturbation.

In these cases it is very difficult to determine how far these undesirable traits can be related to more elementary disorders, and how far these underlying factors may be open to modification. In many cases one finds the association of these undesirable traits, such as stealing, with other evidences of neurotic instability—bad dreams, bed-wetting, masturbation. From the point of view of the criminologist, the material referred to here is extremely important.

The work of the social-service department has been already referred to in connection with other groups, but in no group does the work of this department play such an important rôle, and it is in regard to this group that the responsibility of the dispensary for seeing that advice given is actually carried out, is very direct. Unless the community has already available some efficient organization which is able to deal with the situation in these cases, this duty seems to devolve upon the dispensary, and this work can only be done through a well-organized social-service department. The extent of the problem is considerable, and to have a department which will adequately cope with the situation probably means a very large extension of the work. The results of the work may not appear to be very striking; they are not quickly attained, but they are of far-reaching significance, and permeate deeply the whole social structure. To give an example of the work that is done:

A boy of 11 was being treated at the dispensary for stammering. His home conditions did much to foster the general nervousness, of which his stammering was one expression. The boy was allowed to go to bed

at all hours, usually rushed to school with practically no breakfast, had a rather injudicious diet, and drank coffee *ad lib.* His sisters delighted to irritate him and to provoke outbursts of temper. His mother babyfied him and made him the sport of the other boys. These were facts that were obtained after careful investigation by the social-service worker, and they yielded immediate indications for treatment. The boy now has a regular daily regime; he is in bed at 8.30 p. m.; has a cold tub at 7.30 in the morning, then punches the bag; has breakfast in a sensible manner, with no coffee, and goes to school in time. The sisters have been looked after to a certain extent, and the mother has been encouraged to allow the boy to develop more independently along with the boys of his own age. The boy is fond of manual labor; he has been given a garden in the back of the premises, and at the dispensary he enjoys basket-making. His general speech level has improved, although he still stammers. With regard to his sexual habits the boy is somewhat evasive, but it is hoped that a healthy regime may do much to reduce any difficulties in this sphere.

Now that the boy has been adopted as one of the "gang" which previously used to torment him, his mother, who previously reproached him for his timidity, is alarmed at his newly acquired "toughness," but notices that when he is with his tough comrades there is no trace of stammering.

F. B., a boy of 9, was brought to the dispensary because of screaming spells. He had been a child of slow development, and had a definite speech defect. Whenever he was crossed he got into a violent temper, threw himself upon the ground and screamed until he was black in the face. The parents were dead; his sisters were unable to control him. The boy was nervous and restless at night; he was slovenly in his appearance; he was a truant at school. The home environment was very poor, as the patient and his three sisters slept in one dark bedroom. The social-service worker found that it was possible to put the boy in a new environment; he was placed in the Home for Worthy Boys, where he is looked upon as a very promising child. He is now bright and tidy in appearance; is a regular attendant at school which he enjoys, and shows a very marked improvement in his speech. He comes regularly to report at the dispensary, where he gets speech training.

The mere fact that the abnormalities of a child are taken seriously by the physician, and that the whole subject is investigated thoroughly, has probably a very considerable influence for the better on the home environment of the child. The attitude of the parents becomes fundamentally different.

This was very noticeable in the case of A. A., a boy of 8½, who had been stealing. The father and brother looked upon a thief as a disgrace to the family, and therefore to be relegated to some state institution. To give the boy every chance they brought him to the dispensary to see whether an operation could be done.

The boy was naturally reticent at the first interview. The father did not bring the boy back again, as he did not see that anything was to be gained by simply talking to the boy. The social-service worker investigated the environment, found that the boy was being brought up in the back of a saloon in a colored neighborhood. He was extremely timid, slept badly, was thrown into a panic by a thunder storm. It has been possible to get the boy to come back occasionally to the dispensary, where he makes baskets; he is now on frank terms with the physician. The improvement in the attitude of the father is shown by the fact that on the last episode of pilfering he at once called up the social-service worker on the telephone and asked what was to be done in the situation.

This little episode has really a very profound significance. The work of trying to carry out the treatment, which is essential in such cases, takes up a great deal of time; the individual worker can deal with only a limited number of cases. We feel, however, that the community is responsible for these children, and that this responsibility can only be adequately lived up to when this type of efficient supervision is available for every child who needs it.

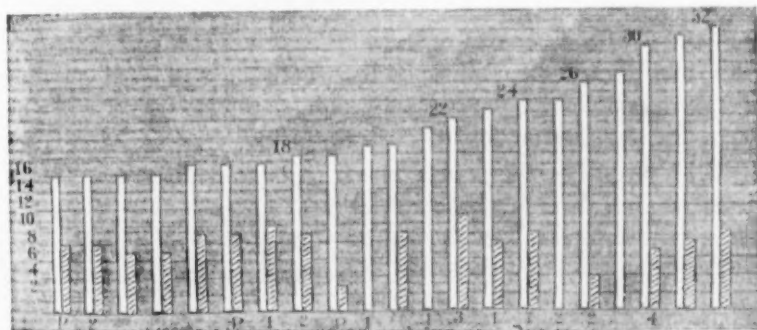
Closely allied in its problems to the group of the abnormal children is the group of adults who presented a marked constitutional defect. This group contained 58 patients; 9 of these showed no marked defect of intelligence, but a marked psychopathic personality which made their adjustment to the environment extremely imperfect; 22 patients showed a marked defect of intelligence with no special complication; 27 patients showed a marked intelligence defect complicated by sexual immorality. Of the 42 women in this group of defective adults, 21 were sexually immoral; the accompanying chart (chart 3) shows the age and mental level of each of these women and the number of their illegitimate children.

The chart indicates facts of very great social importance; it illustrates the future life history of many of the defective children under 16, unless society faces the responsibility of caring for these children. For one with a statistical mind the expense of rearing these children of defectives compared with that of preventing their occurrence would repay study. Another fact that comes out

in this chart is the startling cessation of this material above the age of 32. How is it that above that age the conditions are absent that bring these patients to the dispensary? Have these women succumbed to the terrible risks to which they are exposed? Or have they been disposed of in institutions? Or do they not appear because they no longer become pregnant, nor develop other conditions which bring them to the attention of special organizations?

With regard to the cases of organic brain disorder in our dis-

CHART 3.—PHIPPS PSYCHIATRIC DISPENSARY. MAY 1, 1913, TO APRIL 30, 1914. AGE AND MENTAL LEVEL OF THE 21 DEFECTIVE WOMEN SEXUALLY IMMORAL.



White: Physical age. Shaded: Mental age (B. S. scale). 22 Illegitimate children. P: Pregnant. The numbers beneath the individual cases represent illegitimate children.

pensary material there are few special points to mention. These cases were distributed as follows:

ORGANIC BRAIN DISEASE (58 cases).

*Syphilitic Brain Disease* (29 cases).

General paralysis	15
Cerebral lues	6
General paralysis or cerebral lues	4
Cerebro-spinal lues	3
Tabes dorsalis	1

*Non-syphilitic Brain Disease* (29 cases).

Cerebral arteriosclerosis	14
Senile dementia	5
Brain tumor	2
Brain abscess	1
Double athetosis	1
Unclassified	6



It is worth noting that in the organic conditions, excluding the epileptic and epileptiform conditions, 50 per cent of the patients showed an organic brain disease which was the result of syphilitic infection. It may also be mentioned that the 15 cases of general paralysis in this group already showed a well-marked picture of the disease; they came to the dispensary at a comparatively late period, and not when treatment would have the best chance of yielding results. It will be a great step forward when these patients are brought to the dispensary at the first onset of their symptoms, and it is hoped that as knowledge of these conditions becomes more general these cases will be seen at a very much earlier stage.

Several of the cases of organic brain disease presented interesting neurological symptoms, such as aphasia, and were admitted to the Clinic for the purpose of more accurate study. Several cases were admitted for special treatment.

With regard to the group of schizophrenic states or dementia præcox and allied disorders, it was a rather striking fact that the patients usually presented symptoms for a very long period before they finally came to the dispensary, so that the possibility of modification of the condition by a systematic attempt at the reorganization of the life of the individual was seldom a matter of much promise. Here, too, the general increase of psychiatric knowledge among general practitioners, and the wider recognition among the laity of the functions of the Psychiatric Clinic, will no doubt result in these patients being brought to the dispensary at a very much earlier stage, when the disorder is perhaps more open to modification.

During the year, 78 of the 708 dispensary patients were admitted to the Clinic for the purpose of more detailed study and special treatment.

In reviewing the work of the dispensary during the past year one is impressed with the valuable nature of the material which is there offered for study. It is in the dispensary that one will find perhaps the best material for the study of many nervous and mental disorders in children, and of the incipient stage of many of the disorders of the adult. In connection with the dispensary one will be able to reach some conclusion as to the course which

such disorders run if at an early stage they are taken seriously and an earnest endeavor made to modify those factors which seem largely responsible for the disordered adjustment. After some years one will have material which may be useful in demonstrating that many disorders, which we are accustomed to look upon in a rather fatalistic spirit, can be very much modified by serious treatment, not only of the individual patient, but of the environment in which he is living. The dispensary material emphasizes very strongly how much is gained by considering the individual in relation to his environment, not merely as a unit by himself.

The problem of treatment of the individual case involves a study of the type of personality; of the amount of pliability of the individual; an estimate of the possibility of modifying adjustments of long duration; it involves the study of all those somatic conditions which have much to do with nervous and mental abnormality; it involves the study of the whole situation of the individual's life. The scientific problem is solved when we understand the mechanism of the symptoms in the light of the reaction of the individual to the actual tests put by the environment. The problem of treatment will only adequately be met when we lay due weight on the modification of all the factors which are open to modification, when we are not content with giving advice to the patient based on a detailed analysis of his case, but feel responsible for dealing with the complex situation of which the sickness of the patient is only one aspect. For the satisfactory fulfilment of our responsibilities, however, it is necessary to realize that a somewhat extensive organization will require to be slowly evolved; an organization which should work in close co-operation with other agencies, especially the Committee of Mental Hygiene. An optimist can look forward to the time when it will be possible not merely to say what might be done for a patient, but to see that the actual steps are taken which will give the patient the best chance of returning to an efficient level.



## A REPORT OF FIVE CASES OF THE INTRACRANIAL INJECTION OF AUTO-SERO-SALVARSAN.\*

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It is generally conceded that the so-called parasymphilitic disease, general paralysis, although due, as has been recently shown by Noguchi and others, to specific infection of the central nervous system with the *treponema pallidum*, is seldom, if ever, capable of being influenced through the medium of the blood by any of the antisymphilitic drugs at our disposal.

The reasons for this have been adequately demonstrated in the preceding papers. That these drugs cannot be injected into any part of the central nervous system without the possibility of dangerous results has also been shown. That one of these drugs, viz.: salvarsan, can be so modified as to permit of its injection into the central nervous system has been established by Swift and Ellis of the Rockefeller Institute. Whether such a modification of salvarsan when introduced directly into some portion of the central nervous system is capable of influencing in any way the course of general paresis, it is in part the purpose of this symposium to show. It is unnecessary to describe here the preparation of the Swift-Ellis serum, with which all our cases have been treated.

It is at once apparent that, in a general way, two different methods of applying the serum are open to us. That is to say, it may be introduced by lumbar puncture into the subarachnoid space of the spinal cord, or through a trephine hole in the cranium into some portion of the brain or its meninges.

The intraspinal method has been too well described by Dr. Cotton to need any further elaboration here. Dr. Cotton's results seem to be excellent and distinctly encouraging. At the same time this method in other hands has not always met with such pronounced success. Hough sums up the work of Swift and Ellis of the Rockefeller Institute; Myerson of the Psychopathic Hospital, Boston, Mass., and Asper of Baltimore, as follows:

\* Read at the seventieth annual meeting of the American Medico-Psychological Association, Baltimore, Md., May 26-29, 1914.

There has been marked improvement in the syphilitic inflammatory processes and in many cases the patients with tabes especially, have shown pronounced clinical improvement. Drs. J. A. Cutting and C. W. Mack, of the Agnew State Hospital, California, summarize the result of the treatment of nine cases of general paresis as follows: The most striking result of treatment is the reduction of the cell count, while a review of the mental symptoms is not very encouraging. Dr. Martland and Dr. Beling, of the City Hospital, Newark, have treated six cases of general paresis and cannot report improvement in their condition. We have treated five cases at our hospital by the intraspinal method without improvement in any case. Dr. Edward Mapother and Dr. Thomas Beaton of London have treated four cases without much change in the mental condition.

In view of these rather discouraging reports, and bearing in mind the fact that the disease was incurable, we believed that a more radical procedure was justifiable. That is to say, we believed it was justifiable to inject the remedy into the brain or its meninges. As for the theoretical advantage of intracranial procedures in general the following statement in the *British Medical Journal* by Dr. Harry Campbell is of interest. He says:

The problem, therefore, which confronts us in the treatment of parenchymatous syphilis is how to get the spirillicidal antibodies to enter the perineuronic lymph stream. This end can be gained by intrathecal injections of salvarsanized serum. Whether or not we accept the view of Mott—that the cerebrospinal fluid constitutes the lymph of the central nervous system—certain it is that substances introduced into the subarachnoid space do actually penetrate the central nervous system. If tripan blue be injected through a trephine hole into the cranial subarachnoid space above the tentorium cerebelli, not only the cortex cerebri but the entire cerebrospinal axis is found, post mortem, to be stained. If on the other hand the pigment be injected into the spinal subarachnoid space by means of lumbar puncture the cortex cerebri remains unstained, the staining in these cases being confined to the spinal cord and brain stem. It would appear that the cerebrospinal fluid flows from the cranium spinalwards, probably in meager current, and also that some slight ebb or flow takes place between the fluid in the subarachnoid space at the base of the cranium and the spinal subarachnoid space in accordance with the variations in the quantity of intracranial blood.

It will thus be seen that intrathecal spinal injections afford a ready means of bringing remedial agents into immediate relations with the neurons of the spinal cord and brain stem. While it is probable that the



neurons of the cortex cerebri may in some degree be reached in this way, a far more effectual way of reaching them would undoubtedly be by injecting the cranial subarachnoid space through a trephine hole.

Furthermore, post-mortem injections of carbolfuchsin into spinal subarachnoid space have been made both by us and Dr. Martland of the Newark City Hospital. In no case, however, even with the body placed with head down and in an almost erect position has any staining of the structures beyond the posterior fossa been observed.

Having decided, then, to undertake the intracranial injection of the salvarsanized serum, it was only necessary to cast about for a *modus operandi*.

Four different procedures were open to us: (1) Nisser and Pollac's method of injecting the remedy by means of a small drill introduced beneath the dura through a small area of frozen scalp. (2) Into the subarachnoid space through the orbit, as shown by Beriel, of Lyons. (3) By means of callosal puncture as used by Foerster in Germany and by Dr. Harry Campbell and Mr. Balance in London. (4) The introduction of the remedy subdurally through a trephine hole in the skull, which procedure seems to have been first reported upon by Levaditi, Marie and Martel in December, 1913. It seemed to us that until the value of intracranial injections of salvarsanized serum could be put upon a firm basis that the safest of these procedures, that is to say, the one that was less likely to cause damage to the brain structures, was the one to be experimented with. The first three were set aside as being difficult of application and decidedly risky. The requirement of safety seemed to be best fulfilled by the fourth of these methods, that is to say, by the injection of the serum through a trephine hole in the skull. Accordingly, in conjunction with Dr. Martland, Dr. Eagleton and Dr. Beling, of Newark, we evolved the following operative procedure:

The preparation of the serum is carried out in accordance with the Swift and Ellis technique, viz.: On the day previous to the operation the patient is given an intravenous injection of neo-salvarsan .9 gm.; one hour later about 6 oz. of blood are drawn from the median basilic vein. This blood is set aside to clot at room temperature for about three hours, and is then put in the ice box at a temperature approximating 10°C. until the following

day. The clear supernatant serum is then poured off, centrifuged and mixed with its own bulk of normal saline solution. It is then heated to  $56^{\circ}\text{C}$ . for half an hour and is again placed in the ice box until ready for use. The patient is prepared for operation and given an ether anæsthetic. The trephine hole is bored as nearly as possible over the precentral gyrus. As soon as the dura is exposed a lumbar puncture is performed and about 20 cc. of spinal fluid drawn off. The dura is, as a rule, found rather tense and enough fluid is abstracted to relieve the tension and allow the respiratory fluctuations of the membrane to be plainly seen. When this effect is accomplished the needle is inserted through the dura and about 30 cc. of the previously prepared mixture allowed to flow in by gravity. The apparatus used is an all-glass, 30 cc. Luer syringe, with about 18 inches of rubber tubing attached and an ordinary small caliber salvarsan needle bent upon itself at about a quarter of an inch from the point. After some experience with the cadaver and living subject we find that an opening not less than 2 cm. in diameter is sufficient for the safe introduction of the needle. If the above conditions are complied with the fluid, as far as our experience goes, invariably flows in readily by gravity.

CASE I.—Male; white; 33; married; Pole; tinsmith. Admitted to the hospital June 8, 1912. At this time his wife stated that during the past six months the patient had become nervous, irritable and depressed. He was easily fatigued and had been obliged to give up work. His memory had become so bad that he invariably forgot the errands upon which he was sent. He was exceedingly gluttonous and ate and drank everything he could lay his hands on. He seemed to have lost all interest in his former pursuits and even his affection for his family. Past History: Negative. Venereal: Denied. Habits: To have been good. Physical Examination: Showed a well-developed, somewhat emaciated man. Facial expression vacant. Pupils equal, round, centrally placed and react sluggishly to direct illumination. The facial muscles and the fingers showed a well-marked tremor; the tongue on protrusion a trombone movement. The upper lip was raised with difficulty. Knee jerks were greatly exaggerated. No Babinski, no Oppenheim, no clonus. The gait was shuffling. The co-ordination tests were poorly performed and he had a slight Romberg. Speech showed a marked defect. Otherwise the physical examination was negative. Mentally: He was disoriented as to time, place and person; depressed, forgetful and untidy about his person. No delusions or hallucinations were brought out. The spinal fluid showed excess of globulin and increase of cells. The Wassermann was plus, both in spinal fluid and

blood. He rapidly became more helpless and demented, and on December 12, 1912, he became permanently confined to bed. Examination January 29, 1914: Pupils are equal and regular; do not react to direct or consensual light. Knee jerks are exaggerated, especially on right side. Ankle clonus present; no Babinski. Has well marked apraxia and dysphagia. Cannot walk or stand, or help himself in any way. Is incontinent of urine and faeces. The diet consists entirely of fluids. Apparently understands nothing that is said to him and has not spoken for five or six months. During this interval has had two or three epileptiform convulsions, commencing in the right leg and spreading to the arm and face of the same side. The spinal fluid shows Wassermann 4 plus, 30 cells per cm., and excessive globulin. In the blood the Wassermann is 2 plus. On February 1, 1914, patient received treatment and made an uninterrupted recovery from the operation. On February 5 he was much brighter and made a few purposeful movements. On February 6 he recognized and talked with his wife and answered simple questions as to his physical needs by nods of the head. To date he is still bedridden and incontinent. The apraxia and dysphagia have, to a great extent, disappeared. He talks a little. He is given solid food and feeds himself. The facial expression is much brighter. He can sit up by himself and walk a little with help. On March 12 patient was again treated. On coming out of the anæsthetic the patient had a series of right-sided convulsive seizures similar to those he had had before the first operation. The seizures ceased at the end of 48 hours, leaving the patient with apraxia, aphasia and dysphagia—in short, he was in a condition similar to his status before operation. In a few days these symptoms began to clear up, and by March 29 he spoke occasionally and made purposeful movements. It was decided, nevertheless, to turn back an osteoplastic flap over the site of the second operation. This was accordingly done on April 1 and a small, organized subdural clot was found immediately under the old trephine hole. Following this, the patient improved a little more and at present he sits up, speaks occasionally and feeds himself.

CASE 2.—White; male; 45; married; laundryman; German. Admitted here January 30, 1914. Family History: Negative. Past History: Diseases of childhood, not known. Has never had any severe acute illnesses. Patient was always of a somewhat stolid disposition and had few friends. Was inclined to be irritable and sullen with his wife, but was a good provider. Married at 23. Seventeen years in this country. Both wife and son have syphilis. No history of miscarriages. Habits: Alcohol at times to excess. Venereal: Syphilis at 22. Present Illness: About May, 1913, his wife noticed a change in patient's character. From being rather sullen and stolid he became jovial and expansive, forgetful and somewhat childish in his actions. He became noticeably lacking in judgment, and upon one occasion was arrested for stealing flowers in the cemetery. He finally developed ideas of great wealth and grandeur; could no longer attend to his business and was finally committed to this hospital. Physical Examination: Showed a well-developed, well-nourished man. Complexion muddy, with some cyanosis of hands, feet and middle portion of face. Respiratory

System: Normal. Circulatory System: Normal. Digestive System: Normal. Neuromuscular System: Pupils react sluggishly to direct illumination. Consensual reflex absent on right side. Right pupil somewhat smaller than the left; no irregularity. Knee jerks much increased. No Babinski. Gait somewhat shuffling. No swaying with eyes closed. Co-ordination tests fair. Marked speech defect in ordinary conversation, and patient can't pronounce test phrases. Mental Examination: Oriented for time, place and person. His grasp of the situation and judgment are a good deal impaired. He knows this is a hospital, but thinks there are no insane people here. Says there is nothing the matter with him and seems quite indifferent to his surroundings, making no attempts to explain his presence here. Believes he can go home any time he so desires. Memory for past events is good. Memory for recent events is somewhat impaired. Patient has had a good education, apparently, but school knowledge is, to a great extent, lost. Delusions: He is quite exalted; has great wealth; can speak several languages well. Emotional Tone: He is indifferent to his surroundings, but happy and contented. Speaks of his family without emotion. He has no insight into his condition. Makes many glaring mistakes in calculating figures and displays complete indifference to them when they are brought to his notice. Conduct: He is slovenly in his dress and somewhat untidy.

On February 15, 1914, he received treatment. March 14 patient may be seen sitting about the ward, always occupied in some way. Reads the papers regularly and can quote news items of the day before. Facial expression much brighter. There is none of the indifferent, self-satisfied attitude toward his affairs. On the other hand, he discusses them interestedly and reasonably. Betrays a good deal of emotion when speaking of his family and tells, with pride, of his son's ability and ambitions. There is, however, no euphoria of grandiose ideas. He asks sensible questions about the institution. His speech defect is not so noticeable in ordinary conversation but test phrases are still poorly performed. Physical: The patient has put on weight. His complexion is better and the cyanosis is entirely cleared up. Other signs remain as before. On March 28 he again received treatment. For the last 15 cc. of the serum, slight pressure with the piston was found necessary. On coming out of the anæsthetic it was noticed that the patient was very incoherent and had great difficulty in articulation. This condition persisted for two or three days and then began gradually to clear up. On May 6 an incision was made through the old scar of the left side and a small osteoplastic flap turned back. There was no sign of any hemorrhage. Forty cc. of serum flowed in readily by gravity. At present the patient's status is as follows: He is oriented as to time, place and person and has no delusions of grandeur or wealth. Patient does not understand the nature of his disease or that it is due to syphilis. Thinks, however, that since the operation his head feels clearer and that his memory is better than before. He takes a good deal of interest in his own affairs and wants to have the details of the operation and also of the Wassermann reaction explained to him. Seems to grasp

the outstanding points of the procedure fairly well. Wants to go home, but expresses a willingness to stay here as long as we think desirable. The egotism and feeling of exaltation have given place to stolidity without depression or childishness. There is no aphasia. There is still a marked articulatory disturbance. Other signs remain as before.

February 15, 1914.—Wassermann in blood, 4 plus. Spinal fluid, Wassermann, 4 plus. Cells, 10 per cm. Globulin in excess.

May 1, 1914.—Wassermann in blood, 1 plus. Spinal fluid, Wassermann, 4 plus. Cells, not counted.

CASE 3.—Male; 35; white; laborer (U. S.); married. Admitted February 7, 1914, from Caldwell penitentiary, where he had spent one month of a three months' term, having previously been incarcerated for about two weeks in the Newark jail. The charges against him seem to have been petty thefts and intoxication. Family History: Negative. Past History: Ordinary diseases of childhood. No history of serious illness since. Moderately bright as a child. Can read and write and add figures, but his education extends no further than this. Always a good workman. No previous mental abnormalities noted. Habits: Moderately alcoholic, occasionally drunk. Of late has drunk to excess. Venereal: Gonorrhea denied. Syphilis probably eight years ago. Present Illness: About one year ago patient was noticed to be irritable and faultfinding, with both grandiose and persecutory ideas. At times threatened to kill members of the family. Frequently drunk. Went away from home and lived with a woman whom he claimed as his wife, although there is no record of marriage. Came home after a while intoxicated, and stole a few small articles, for which his sister had him arrested, and he was given three months at Caldwell penitentiary. Summary of Condition at Caldwell Penitentiary: The patient was very restless in his cell and destructive, so that he had to be put in a padded cell. He managed to find a loose spot in the padding and tore it away, doing much damage to the cell. When taken to the bath room for a bath he went at once under the spray with all his clothes on. Mental Condition: The patient is very destructive and excited; uses alcohol to excess; sleeps irregularly; eats well. Said he intended to establish a printing office next week and would raise chickens and vegetables in conjunction with the business and thus make a large amount of money. Said he had never lost a position. He tore up his sheets and blankets to make handkerchiefs; claims he could make use of the pieces. Excited at first, but later laid down on a bare board with his cap for a pillow and said it was a very comfortable position. On admission here he was excited and restless, tearing his bedding and climbing up on the window guards. Talking incoherently. Physical: Shows a well developed man, very much emaciated. Complexion sallow. Expression vacant. Respiratory System: Normal. Circulatory System: Some cyanosis of extremities. No other abnormalities. Blood pressure, 120. Neuromuscular System: Eye movements normal. Pupils equal; react sluggishly to direct illumination. Do not hold well. Coarse tremor with hands extended. Coarse tremor of tongue. Knee jerks and



ankle jerks, increased. No Babinski, no Oppenheim, no clonus. Gait somewhat shuffling. Station good. Patient performs the ordinary co-ordination tests rather poorly and is unable to button his coat. Sensation: Patient is generally hypoæsthetic. Does not wince when lumbar puncture is performed. Taste and smell normal. Speech low, monotonous and thick. He can't repeat test words. Cannot write his name and address. Mental Examination: Orientation; gives his name correctly. Does not know the name of this hospital, although he recognizes its general character. Gives several contradictory answers as to where he came from and why sent here. General Memory: Gives confused and contradictory answers as to his past life, members of family, etc. Is 35 years old (correct); was born in 1865; school knowledge very poor; 8x9 are 34. California is in New Orleans. Can't give capital of country or state. Delusions are grandiose in character, very exalted. Has \$60,000, automobiles, horses and stores. Has lots of friends. Is very good-looking and well-built. Much stronger than the average man. Can drink 50 glasses of whiskey a day. Insight: None. Is here because he has a pain over his kidneys. Is mentally sound. Is not at all affected when his numerous mistakes and shortcomings are brought to his notice. On March 6, 1914, patient received treatment. March 16: Patient is working actively about the ward. Energetic and interested in his surroundings. Facial expression is greatly improved. Complexion ruddy and healthy in appearance. Other physical signs unchanged. Patient is oriented for time, place and person. He knows how long he has been here and gives a correct account of his experiences immediately preceding his coming here. Still insists that he was married. He is somewhat exalted and slightly euphoric. Denies having any large amount of property now, but has no doubt of his ability to secure it and is full of plans for the future. He has \$6000 in the bank and he is going to start a garage, also buy out his old employer in the printing business. This printing business is worth only about \$2000, he says, and his ideas as to the garage are not exceedingly extravagant. There is a well-marked increase in cutaneous sensibility to touch and pain. March 24 patient again received treatment. April 2, 1914: Patient continues to gain physically and mentally. Delusions of grandeur entirely disappeared. Has no doubt as to his ability to take up his old work. Emotional reaction normal, and patient appears sane in every way. Remembers some of the things he said and thinks he must have been crazy to express such ideas. Gives a clear, connected account of his past life. His reaction toward his environment is normal. His judgment is good, and although his education is not such as to allow his understanding the exact nature of his illness, he realizes that he has had some mental trouble, and is willing to continue treatment.

February 28, 1914: Wassermann in blood, 4 plus. Spinal fluid, Wassermann, 4 plus. Cells, 109 per cm. Globulin in excess.

May 1, 1914: Wassermann in blood, 2 plus. Spinal fluid, Wassermann, 4 plus. Cells, 33 per cm. Globulin unchanged.

CASE 4.—Male; white; 47; married (U. S.); proofreader. Family History: Father died of apoplexy. Past History: Ordinary diseases of

childhood; no severe acute illnesses. Always of a nervous disposition. A very hard worker, always working 12 to 14 hours a day. Held a very good position as head proofreader on *Collier's Weekly*. Habits: Alcohol to excess at times, but never lost a day's work through intemperance. Venereal: Denied. Present History: He began to act peculiarly in August, 1912. Was somewhat incoherent in conversation; would leave his work and go home before the specified time. Was sent to the country to recuperate, but did not improve. Was continually depressed. Walked about the house talking to himself and muttering words about his wife. Was extremely forgetful. Had delusions of grandeur. Admitted to this hospital October 21, 1912. Physical Examination: Shows a well-developed poorly nourished man. Respiratory System: Negative. Circulatory System: Negative. Digestive System: Negative. Neuromuscular System: Pupils react sluggishly. Knee jerks markedly exaggerated. Coarse tremor of tongue and fingers. Tremor of facial muscles. No Babinski. Sways slightly with eyes closed. Co-ordination tests poorly performed. Marked speech defect in ordinary conversation. Can't pronounce test phrases. Mental Examination: Is disoriented as to time and place. Memory for past events, fair. Memory for recent events, very much disturbed. Delusions: Imagines that all his organs are weakened; thinks his bowels never move. Thinks this is a hotel and that it belongs to him; that the whole world belongs to him; thinks he has millions of dollars, thousands of elephants, tigers and lions, and a harem full of giant wives. Emotional Tone: Is extremely irritable, unapproachable and easily enraged. Noisy and abusive. Restless and continually annoying the patients. Talks almost incessantly and frequently breaks out into fits of obscene fury. Tears up his clothing and bedclothing. Smears urine and feces about the room. May 29, 1913: Patient had three convulsions. August 9, 1913: Shouting continually. Is delusional, incoherent, has exalted ideas. February 11, 1914: Is in a continuous noisy state; talks and sings all day long; is quite unapproachable; seem to have memory for recent events; thinks his mother is dead, although she visited him the day before. On March 7, 1914, patient received treatment. Patient's condition remained about the same for about a week, continually talking, shouting and singing day and night. At the end of this time he gradually began to grow quiet; slept well at night and during the day had fewer outbursts of rage. On March 27 he again received treatment. Following this operation, improvement was continuous. At the present time he has taken on a good deal of weight, his complexion is ruddy and healthy in appearance. From being practically bedridden, he is able to take daily walks about the premises. His attitude has changed decidedly. Although not decidedly friendly, he is quite approachable. He is never noisy except when purposely annoyed. He is not destructive and he is tidy about his person. The speech defect is not so noticeable. His memory for recent events is much improved. He is still disoriented and the delusions of grandeur persist.

November 10, 1913.—Wassermann in blood, 2 plus. Spinal fluid, Wassermann, 4 plus. Cells, 50 per cm. Globulin in excess.

April 14, 1914.—Wassermann in blood, minus. Spinal fluid, Wassermann, 4 plus. Cells, 3.5 per cm. Globulin in excess.

CASE 5.—Male; white; 35; married (U. S.); dentist. Admitted August 28, 1913. Family History: Negative. Past History: Ordinary diseases of childhood, no severe acute illnesses. Said to have been one of the best dentists in Newark. No mental abnormalities noted until present illness. Habits: Moderate alcohol. Venereal: Syphilis 12 or 15 years ago. Present Illness: About four years ago patient's wife began to notice a change in her husband's character. He would buy and bring into the house all kinds of unnecessary articles, especially tools and instruments of various kinds. He also went into foolish real estate ventures. Finally it became necessary to have him committed. On admission here physical examination showed a well-developed, well-nourished man. Respiratory System: Normal. Circulatory System: Normal. Digestive System: Normal. Neuromuscular System: Pupils normal and react fairly to light. Slight tremor of tongue and fingers. Knee jerks almost absent. Co-ordination tests fairly well performed. Walks straight line with some difficulty. No Romberg or Babinski. Somewhat hypoæsthetic. Marked speech defect in ordinary conversation. Test phrases cannot be pronounced. Mental Examination: Oriented as to time, place and person. Memory for past events somewhat confused. Says he is 36 years of age and has been working at dentistry for the past 22 years. Cannot do simple sums in arithmetic. Cannot answer simple geographical or historical questions. Emotional Tone: He is happy and contented; somewhat exalted. Has no insight into his condition, and shows complete indifference when his numerous glaring mistakes are pointed out to him. Has no special delusions. On March 15 patient received treatment. There has been no special change in the patient's condition since he has been in this hospital, either before or since operation.

March 1, 1914.—Wassermann in blood, 2 plus. Spinal fluid, Wassermann, 4 plus. Cells, 3.3 per cm. Globulin in excess.

CASE 6.—Male; white; 33 (U. S.); married; salesman. Admitted first, May 9, 1913. Family History: Mother and maternal aunt insane. Past History: Diseases of childhood, mumps and measles. He was bright in school; common school education, after which he went into the electrical supply business, where he proved himself a good salesman and drew good wages. Married at 20, and has four children, all alive and well except a baby, who died of measles three weeks before his coming here. Has never showed any mental abnormalities. Habits: Moderately alcoholic; never drunk. Venereal: Syphilis nine years ago. On his first commitment here he was said to have been acting in a peculiar manner for about six weeks. He was exalted, excited, with grandiose ideas; he was worth millions and had affairs of the utmost importance to attend to. Physical Examination: Showed a man in fair state of nutrition. Eyes, lateral and verticle nystagmus; right convergent strabismus. Pupils react sluggishly to direct and consensual light. Deep reflexes increased. Gait and station

good. Some tremor of fingers and tongue. No other abnormalities. The patient remained in the condition described for about three months, becoming much emaciated. At the end of this time he began gradually to improve mentally and physically, and on September 11, 1913, he was taken out against advice. He went back to his home and business and seems to have got along fairly well, although according to the statement of relatives he was not entirely normal mentally.

On March 17, 1914, he was suddenly taken with convulsive seizures, which lasted two days. He was removed to the City Hospital, where he became rapidly much deteriorated and helpless. On admission here on April 8, 1914, he presented the following picture: Patient lies in bed with dull, expressionless facies. Conjunctiva glassy; eyes vacant; sordes on teeth and lips; tongue cracked, brown-coated and dry. Decubitus on left heel about the size of a silver dollar. Complexion sallow. Fairly well nourished. Circulatory System: Heart appears normal in size and position. No murmurs. Pulse rapid, small, not well-sustained. Hands and feet somewhat cyanosed. Respiratory System: Normal. Abdomen: Normal. Neuromuscular System: Pupils equal in size; react sluggishly to direct illumination, the left more so than the right. The left pupil does not react to indirect illumination; the right only slightly. Tongue shows coarse tremor. Tremor of fingers. Knee jerks exaggerated. No Oppenheim, no Babinski. Has some difficulty in distinguishing between the dull and sharp end of pin. Cannot stand or walk alone. Could not feed or help himself in any way. Would not turn himself over in bed. Mental Examination: Patient's attention can be momentarily attracted by talking to him, and he gives his name. Is unable to give date, day, or name of hospital. Can answer no questions of any kind. On April 9 patient received treatment. Grasp: Gives date. Remembered only in a very vague way that he was at the City Hospital, and that he was brought here and operated upon. Has a confused memory of sitting in a chair and having his head shaved; of being given an anæsthetic and waking up with a bandage on his head. His recollection of this period and for several days after is very fleeting. The first event he remembers with distinctness, and from which accurate memory has continued, was being allowed to put on his clothes, which he was allowed to do on April 13. Understands in a general way the nature of his illness, and is anxious to continue treatment. Says he feels in bad shape. Patient's speech is low and rather thick, but there is no elision of syllables, and test words are fairly well repeated. Patient has since received a second treatment and has continued to improve. He is completely oriented. Memory for past and recent events, excepting his stay at the City Hospital, is good. He has no delusions of any kind. His reaction to his environment is in every way normal, and he has good insight into his condition. Physically he is much improved. He takes extended walks about the place and has put on weight. Test phrases still show speech defect, but in conversation there is none.

April 1, 1914.—Wassermann in blood, 4 plus. Spinal fluid, Wassermann, 4 plus. Cells, 80 per cm. Globulin in excess.

May 1, 1914.—Wassermann in blood, 3 plus. Spinal fluid, Wassermann, 2 plus. Cells, 45 per cm. Globulin unchanged.

From a clinical standpoint two cases, 3 and 6, have had complete remissions after two treatments. One has remained unchanged for two months and the other for one month.

Case 3 has had three treatments, and clinically has a remission with slight impairment of apperception and judgment.

Cases 1 and 4 have had three and two treatments respectively and have shown marked improvement, lasting in the first for three months and in the fourth for two months.

Case 5 has had one treatment and has shown no change. Two other cases have been operated upon so recently as to preclude a report on their condition at present.

The Wassermann reaction in the blood has, in all cases, been reduced in intensity and rendered negative in two cases. In only one case has it been reduced in the spinal fluid, where it was brought from 4 plus to 2 plus. The globulin reaction has remained unchanged. The cell count has been substantially reduced in all cases.

The cases treated were not specially selected but were taken at random, the patients being in all stages of the disease. The best results were obtained in those cases in which the first manifestations of the process have been noticed within a year or less, and in which the actual destruction of nerve tissue might reasonably be supposed to be comparatively slight. Conclusions as to the absolute and relative value of the treatment are as yet somewhat difficult to draw. The series of cases is still small and the period of observation short. We believe, however, that our results have been sufficiently striking to warrant further investigation along this line. We believe also, and we think special stress should be laid upon this point, that the treatment is in no sense heroic, and when properly undertaken is practically without danger. It consumes little time and is well borne by the patients in all our cases. We think it possible that an ideal treatment of general paresis may eventually be found to consist in a judicious combination of the intracranial and intraspinal methods.



## INSANITY IN CHILDREN.\*

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This presentation deals with mental disease in children exclusive of choreic, epileptic and hysterical insanities, juvenile paresis, syphilitic dementia, and the idiocies. Dementia precox is also excluded, as the cases to be referred to, in the main, concern children under 15 years of age.

At first sight insanity in children would appear to be a rare affection, indeed Hagen<sup>1</sup> stated that it occurs in one in every 72,752 inhabitants only. Ireland believed that it was so uncommon as to make it very difficult to generalize about it. But the study of the literature forces one soon to the conclusion that there is "nothing new under the sun" after all, for Coelius Aurelianus as long as 1400 years ago described mania in children. The earliest recorded case since then is that by Berkham<sup>2</sup> who in 1750 observed a case of melancholia in a girl of eleven. Greeding<sup>3</sup> in 1790 also published a case of insane fury in an infant of 9 months of age, and in 1794 Perfect quoted by Gottgetren<sup>4</sup> described a case of depression followed by mania in a lad of eleven.

Delasiauve in 1852 was the first to describe mania in children according to Moreau,<sup>5</sup> who himself stated that among the mental affections of childhood mania was the first to which the attention of the authorities had been called.

I have studied exhaustively the literature of insanities in children and have collected 44 cases which seemed to be worthy of citation. Considering the importance of the subject there has been less consideration given to the study of psychosis in childhood than the subject deserves.

The rareness of youthful cases in asylums is well known. This is probably due to the fact that the children afflicted with psychoses

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are more readily treated at home than adults, for the reason that the duration of the attack is shorter than in adults, as well as for the fact that the symptoms are less severe.

A study of the cases reported by me and quoted from the literature will show that, outside of a few cases of post-infectious mental states, the usual type of mental affections in children is the manic-depressive form of insanity. While in my own experience, the usual form of psychosis is the manic type of manic-depressive insanity, of thirty two cases collected from the literature thirteen were of the circular form, eleven of the manic form and eight of the melancholic type. The circular type or, as it has been termed by Baillarger<sup>6</sup> in 1854, folie à double form, was described by Lunier<sup>7</sup> and others, and was recognized by Moreau in his work in 1888. Melancholia has generally been supposed to be rare in children. Eminghaus<sup>8</sup> had never seen an instance of periodic melancholia, though in 199 cases of insanity in children there were 24 cases of melancholia.

Besides these types of insanities, there are a number of cases, fairly common in my experience, of psychasthenia, in which the affection is characterized by mental instability, depression and excitability associated with phobias and hypochondriasis.

The infectious psychoses are well known, though I do not believe are of common occurrence. I found only twelve cases quoted in literature after a careful search. Spitzka<sup>9</sup> found 7 per cent of his cases sequellæ to fevers. The post-febrile insanities of children, according to Ireland, are often found associated with hereditary predisposition.

Alexander<sup>10</sup> believed that 10 to 30 per cent of the infantile insanities were attributable to the acute diseases of childhood, especially the exanthemata.

Cases of insanity in children have been described following scarlatina (Ireland,<sup>11</sup> Welt,<sup>12</sup> Gerlach,<sup>13</sup> Joachim<sup>14</sup>), diphtheria (Kuhn<sup>15</sup>), pertussis (Mischede<sup>16</sup>), typhoid fever (Welt, Jensen,<sup>17</sup> Jordan,<sup>18</sup> Feith,<sup>19</sup> Nierier<sup>20</sup>) and a cold during malaria (Hume<sup>21</sup>). In six of these cases hallucinations were observed, six were excited and agitated, one was depressed and had obsessions and one exhibited a stuporous state.

Except in the post-infectious insanities, hallucinations and delusions are not common, the symptoms consisting of either simple

depression or excitement. There were seven cases that exhibited hallucinations, two of which were cases of melancholia and only three were delusional, and one of these a case of melancholia. Systematised delusions do not occur, according to Kraft-Ebing,<sup>17</sup> and Ireland stated that fixed ideas and delusions are not common. The older the patient, or the more nearly he reaches 16, the more likely is he to become the subject of hallucinations and delusions for obvious reasons.

The youngest case of insanity in children on record is nine months. Of forty-seven cases collected by Berkham<sup>18</sup> there were 10 cases under 5 years of age, and most of the remainder occurred between 10 and 11 years of age.

Of thirty-nine cases collected from the literature by myself, including my own seven cases and exclusive of the infectious psychoses, there were fourteen under 10 years of age and twenty-five between 10 and 16.

CLASSIFICATION OF CASES AS TO AGE OF ONSET.

5 years.....2	11 years.....4
6 " .....2	12 " .....1
7 " .....1	13 " .....9
8 " .....1	14 " .....3
9 " .....3	15 " .....5
10 " .....5	16 " .....3

An interesting question arises in the study of these cases: What bearing has insanities during childhood upon the future mental health of the individual during adult life? It is to be noted that all the cases recover from the attack; few with any perceptible mental damage. We may well ask whether these attacks are not the forerunners of more serious attacks in later life. A case has only recently come under my observation which throws some light upon this question. A girl of 15 had obsessions at 3½ consisting of an impulse to kiss soiled objects, such as dirty boots and the seat of the water closet. From this she recovered and at 6 developed a psychosis after tonsilitis which lasted nine weeks. At 15 she lost interest in things, did things less well than formerly, was careless, became cross, unstable, depressed, readily fatigued, sleeping poorly and was apathetic. These symptoms are danger signals in a case with a psychopathic history and it is just this sort of a case which

needs wise care in order to prevent a recurrence of a psychosis.

The first case to be reported is interesting not so much by reason of the symptoms he presented, but on account of the fact that periods of excitement were regularly followed by a depressive state, usually with a period intervening in which the boy was perfectly normal, therefore being an example of the circular type of manic-depressive insanity.

George V., A boy of 15. Healthy parents. A grandmother's cousin died insane, and a second cousin was insane at the time of his death. There is no history of other mental or nervous disease or of tuberculosis or cancer.

George, the patient, had always been healthy except for an attack of diphtheria at 8 years of age and two attacks of erysipelas three years previously, and an attack of measles in childhood.

He was what his mother termed a sensitive child and his feelings were always to be considered a good deal. He was a good boy, was companionable and was said to be a leader among his playmates. On account of the attacks of erysipelas he had lost time at school and in the spring of 1912 he was pushed in his studies in order to pass his examination in the eighth grade grammar school.

In July, 1912, his mother stated that he began "to have something on his mind." He was very depressed, self-accusatory and considered himself very wicked. He practiced masturbation and was very depressed over this, attributing his condition to this habit. He went to bed for three weeks, complaining of insomnia, of general languor, and aching of his limbs and body. He worried continually over the wrong he had done and believed that he would never get well. At the end of five weeks these symptoms cleared up and he seemed to be normal for about a week, whereupon the symptoms recurred, and he went to bed again. During this attack he was circumcised and took osteopathic treatment.

At the end of two weeks he became exhilarated, thought of and talked about wild Indians and the things he had seen in moving picture shows, wished to buy guns and to run to stores. He began to steal things not especially useful to him or for which he had any special need, acknowledging the thefts and giving no satisfactory explanation of the act. He was in this attack very active, talking a great deal and distinctly excited. At the end of a week these symptoms subsided and a period of depression occurred which lasted from the middle of September to November 1. He then again became excited, packed his clothes and said he was going to leave home because the family could not trust him. He intended to become a hermit. This phase lasted two weeks, to be replaced again by a period of depression. It was while in this condition he came under my care on November 24, 1912. At that time he was depressed, looked unhappy, confessed to masturbation, which he considered a great sin and responsible for his illness; believed he was wicked and thought he could not recover.

He was readily fatigued, complained of general aches and persistent occipital pain.

His memory was poor; he said he did not know clearly what he was doing and had difficulty in concentrating. He was underweight, pale, his appetite was poor, his tongue was coated and tremulous. He complained of indigestion and was constipated. The examination of his heart and lungs was negative. The knee jerks were sluggish, his station good, his pupillary reactions normal. The dynamometer registered 46 both on the right and left. Urinary analysis revealed nothing abnormal.

He remained depressed from November 24 until December 5, when he became again excited. He sang or talked constantly, was exhilarated, happy, careless, purloined things from the closets and the nurses' bags, knocked on the doors of adjoining rooms, kicked around in bed, pounded on walls and wrote obscene letters. This period lasted 18 days, to be replaced by depression for 10 days. There then followed an interval in which he was perfectly normal for 14 days.

Periods of depression lasting about 10 days were followed by periods of excitement of the same duration, a period intervening of about a week to 10 days, in which he appeared perfectly normal.

Under treatment he improved, the symptoms during the attacks becoming less marked, and finally in June, 1913, he became normal most of the time and remained so for a few months. I have learned recently however that the symptoms returned. In the last 7 weeks he appeared to be normal again; is at a military school and from all reports is doing well.

The second case illustrates the manic form of manic-depressive insanity, with tendency to recurrences.

Joseph L., aged 9, applied for treatment January 25, 1905. There was no history of nervous diseases or insanity in his ancestors. The patient himself had measles at 3, typhoid fever at 4, and whooping-cough at 6.

Joseph was always bright, acquired readily, was fond of reading, was really precocious in his literary tastes, devouring Shakespeare's plays voraciously at the age of 9. He was, however, always somewhat irritable.

In November, 1904, two months before coming under my care, his teacher asked his mother to take him from school because of his bad temper, which was provoked by any attempt at discipline. On one occasion he took off his shoe and attempted to beat his teacher. When I first saw him in January, 1905, he was anemic and somewhat under weight. His physical examination was otherwise practically negative. The reflexes were normal; there were no sensory or motor troubles, and his pupillary reactions were normal. He was astigmatic and hypermetropic, but the eye grounds showed nothing amiss. His memory was good; there were no delusions or hallucinations; he was perfectly oriented and he concentrated well. His expression was rather excited, his eyes were shifting and he was physically restless. He was also somewhat resistive and it was necessary to exercise considerable care in talking to him in order to avoid arousing his antagonism.



onism. His mother informed me that he grew tired readily and that he was always more difficult to control at the end of the day; also that he was very quick tempered and had always to be handled with great care. I took him from school, advised rest, baths and increased amount of food and tonics, with the result that considerable improvement in his condition ensued. He was very much improved at the end of three months. He remained fairly well until June, 1906, although during this interval of a year there were occasional lapses, consisting of violent outbreaks at the end of the day, usually connected with some incident relating to his mother, but most of the time he was controlled.

In June, 1906, he became very excited, having periods every day of excitement, in which he would become profane, throw pillows and bolsters downstairs in anger and seemed to be in a very high temper. This subsided at the end of a few days, but there were recurrent similar attacks until November, 1906. From then until Christmas, 1906, he was in a very good condition. Then, however, he began to be excited again and was violent, rolled around on the floor, was bad tempered and profane. These symptoms persisted and on February 7, 1906, he got into an altercation with a man and boy on the street and afterwards he became very excited, cursed and complained to God for causing his condition. He said "God does not give me any show." "Why should he punish me so?" "I would be a great deal better dead." I sent a nurse to him, and gave him a partial rest cure. At first he was very violent, very abusive, and threatened to kill his mother and himself. He said to his mother, "You ought to go up an alley with a man," and made similar obscene statements. He was very resistive, refusing food, medicines and treatment. He attempted to choke his mother on many occasions, kicked his nurse repeatedly, rolled around in bed and swore at her. This attack lasted about two months, at the end of which time he gradually improved.

He returned to school in September of that year and remained at school until one year ago, having spent two years in the West Philadelphia High School.

Since a year ago he has taken two or three positions as a clerk. He did not care much for the work, but said he preferred the life of a teacher or a newspaper man. He was apparently normal until July, 1913, when he awoke with a headache one morning and remained out of work that day. His mother dates his present attack from this time. Joseph himself believes that the present attack began on the 6th of August, 1913, although he remained at work until the 14th of September, since which time he has not worked. He states that he has been fighting the thing off ever since the 6th of August. He could not keep on with work because he "got all balled up." He was quiet, somewhat depressed during the autumn and he showed no violence until January 1, when he broke a sink when irritated over some clothes that he had bought that did not fit him. Four days later he refused to get up in the morning, kicked the walls of his room, swore and shouted out loudly, calling his mother names and swearing at her. On the 8th of January, at 4 p. m., suddenly, without provocation, he said to his mother,

"Damn you," and broke a flower pot, then ran upstairs and broke things up. He later asked for coffee and said, "Make it damn quick."

In the evening started to go out, saying to his family they would never see him again. In trying to restrain him from going out a struggle ensued, in which he smashed a couple of doors and engaged in a battle with his brother. Officers were summoned and he fought them, but was finally subdued and taken to the station-house, where the police surgeon communicated with me and by my advice he was sent to the detention ward at Blockley. He was transferred from Blockley to the Pennsylvania Hospital for the Insane at the end of two weeks time, where he has remained ever since. Since his admission he has been very quiet and orderly. He does not recognize his condition or that he behaved in an abnormal manner, or at least does not admit it.

Katharine G., aged 16, illustrates the manic type. She has a colored father and a white mother, both of whom were well. The family history is negative. There is no history of insanity or nervous diseases. Katharine had the usual diseases of childhood, menstruated at thirteen and was regular. Her present trouble began shortly before her visit to my clinic at the Polyclinic Hospital July 10, 1911. She became restless, slept poorly, dreamed a good deal and was very absentminded. She was disorderly, frequently arose at 3 a. m. and went out of the house, or during the day wandered into other people's houses. She was very cross and disobedient. She heard people talking about her and conversed with the voices. She made fists and struck at people. She believed that the neighbors called her all sorts of names, and she wanted to fight them. Their house was the only decent home in the court.

Katharine was a girl of average height and development. There were no palsies, the tongue protruded straight and in the median line, the station was good and the knee jerks were equal and normal. The examination of the heart and lungs was negative. She gradually improved and at the end of a month the hallucinations and delusions had entirely disappeared. She was still well a year later, at which time she had been working for some time.

The fourth case is one of the manic type.

Vincent W., aged 12, was admitted to the Philadelphia Polyclinic Hospital November 30, 1911. Both parents were living. The father had had a chancre and the mother had had two miscarriages. Two brothers died in convulsions about one year of age, but otherwise the family history was negative. Vincent was born at term without instruments. When three years old he fell from a shed and struck the back of his head, but was not rendered unconscious. At the age of 8 he had a convulsion, but none since.

About a month before his admission he began playing truant, was inattentive at school, and silly in his conduct. He would twitch his face and draw on his desk. He was constantly thinking of cowboys and Indians suggested by moving pictures. He played in a tent in the yard constantly

and decked himself like a cowboy. He hid himself in the cellar and was found lying prone on the ground, apparently deep in some cowboy adventure. He was seen to draw a sharp knife over his sleeping sister. His expression was wild, his eyes restless and staring. It was difficult to hold his attention and he was physically very restless, thrashing around in bed and was kept in bed with some difficulty. He complained of frontal headaches associated with some nausea, dating since the onset of his mental symptoms. His appetite was good and his bowels regular. He was thin and anemic, but slept well. The station was good. The pupils which were widely dilated reacted normally. The extraocular movements were intact and he had hypermetropic astigmatism. The knee jerks were normal, and there was no ankle clonus or Babinski phenomenon. The examination of the heart and lungs showed no abnormality. Vincent gradually recovered and was discharged cured at the end of a month.

Samuel S., a Hungarian, aged 16, illustrates a mild manic type. He came to the clinic of the Polyclinic Hospital, April 12, 1912. There was no history of insanity or nervous diseases. He had had measles, but otherwise had not been sick since birth. He went to school until a year previously and had done well.

His present trouble began three weeks before his visit to the hospital. He awoke excited in the middle of the night and thought that the house was on fire and that a man was robbing the house. The next day he was confused in his speech, was difficult to control and was excited. Upon examination he was confused and incoherent. His replies to simple questions consisted of merely articulating a letter of the alphabet. For example, when asked where he lived his reply was "M." He could not sleep and wandered around in an excited manner.

The physical condition was fairly good but he was somewhat thin and anemic. The tongue was protruded straight and in the medium line and was coated. His hands were cyanotic and cold. The station was good and the knee jerks were much increased and equally so on both sides. The physical examination was otherwise negative. He was admitted to the wards, but was so disturbed that it was impossible to take care of him on account of his excited condition and he was taken to his home the next day. Under treatment at the end of three months his symptoms disappeared and he was able to go to work. He was still well at the end of nine months, since which time I have not seen him.

W. N., aged 13, applied to the Philadelphia Polyclinic Hospital April 13, 1914. This case is an example of transient brief maniacal attacks. The father had had an apoplectic stroke; was living and was not alcoholic. The mother was asthmatic. There was no epilepsy, tuberculosis or insanity in the family. William had six brothers and a sister living and well. He had had convulsions when a baby and chorea three years ago. He was bad tempered as a child. He was perfectly well until four weeks before applying for treatment, when he began to act strangely. There was first a feeling of sleepiness and a sensation as if the blood had rushed to his

head and he acted as if in a temper. He remained at school until two weeks ago. Five days previous to his first visit he fell unconscious and frothed at the mouth. Three days later he became very excited, talked loudly and swore. He used obscene language, threw things at the members of the family, broke up furniture, threw knives about, and tried to jump from the window. This condition appeared without provocation and lasted for about two or three hours. The following day he had a second attack of a similar nature. The family said that in the interim he did not want to go out to play as usual and sat with his head bowed forward and was very quiet and talked very little. He said to his family that he wanted to run away and drown himself.

Upon examination he stated that he was very unhappy. No delusions or hallucinations could be elicited and he was perfectly oriented. He had, however, no memory of the attacks of excitement. He was mentally sluggish and unable to describe his sensations or his feelings. He complained often of vertical headache and slept poorly, sometimes remaining awake all night. His tongue was slightly coated and teeth indented. His pupils were equal and reacted to light and inaccommodation. His hands were cold and somewhat cyanotic. His pulse rate was 120 and his heart sounds were perfectly clear, but the tension of the pulse was high to the finger touch. The knee jerks were increased, but equal on both sides. His face had a somewhat anxious expression and was pallid. I admitted him to the wards and placed him on the rest treatment with bath, massage, and forced feeding. He improved rapidly, became cheerful and alert, and was discharged to the out-clinic at the end of three weeks. I saw him a few days ago, and he was still well.

#### MELANCHOLIC TYPE.

E. W. is one of two cases of pure melancholia in my series. She was 11 years old when she applied to the Neurological Department at the St. Agnes Hospital on February 17, 1898. Her father was subject to nervous headaches and her mother was living and well. There were five other children, all of whom had had spasms, except one, during teething. One of her brothers at the time of his death was epileptic and insane. The patient herself was a 9-month child and was born without accident; was breast-fed, walked at 12 months and talked at 14 months. She had had scarlet fever eight years previously. Her chief complaint at the time of applying for treatment was epileptiform attacks, dating two years previously. She has had one convulsion at 2 years of age and one again at 4. Since November, 1896, she has had five or six grand mal attacks a year with numerous petit mal attacks.

Her physical condition was fairly good. Her tongue was coated; her teeth were good and her palatal arch was high. The knee jerks were exaggerated and equal on both sides. The station was good. The heart and lungs were normal. She had no scars on her head and there was no history of her having had an accident. An examination showed absence of

any source of reflex irritation in her nose and her vision was properly corrected.

She was clever at school and was normal, cheerful and good-natured.

In 1898, when she was 12 years old, while listening to instruction for her first communion she became very much depressed. She was very unhappy; felt that she had been guilty of committing many sins. Every act she interpreted as a sin. The depression was very much worse upon awakening. She was much absorbed by these depressive thoughts and did not recognize at the time that they were delusions. These symptoms lasted about a month, when the depression cleared up entirely. Incidentally the epileptic attacks diminished gradually in frequency and in 1904 ceased entirely.

Morris F., aged 13, the second case of melancholia, applied to the Polyclinic Hospital February 16, 1914. Family history was negative. There had been no history of insanity or epilepsy or other nervous diseases. The patient had had measles, but had otherwise enjoyed good health. He was clever in his school work, having reached the Fifth Grade A.

His present illness began in June, 1913, when he was struck by a baseball over the left eye. He was not rendered unconscious and was able to leave the hospital in a day. He has been nervous and restless ever since. His father stated that he acted in a stupid manner; he slept very poorly; complained of headache, especially at night, and in the three weeks prior to his going to the hospital had dizzy spells. Two months ago he fell unconscious to the floor, but did not have any convulsion. Recently he became very much depressed, refused his food and did not play with his companions, sitting around the house all day long, voluntarily making no remark to anyone.

At the time of the examination he was apparently very much depressed. His face was expressionless, his eyes were fixed in one direction and he could be aroused from his revery only with great difficulty. He denied hallucinations of hearing. He did not know why he was depressed or what he was worrying about. He mumbled his replies to the questions of the examiner, blinking his eyes and giving indefinite replies. He never smiled and said that he felt sad and was sick at heart. At times he would walk constantly around the house without any object. This patient is still under observation, having improved very considerably, though he had not entirely recovered when seen last.

In a thorough search of the literature available I collected forty-four cases which seemed to have been recorded sufficiently carefully to warrant quoting. These cases can be divided into the manic-depressive type, including the circular, manic, and melancholic types, post-infectious psychoses, and one case interesting as illustrating a precox type of psychosis which is not readily classed. These cases have been described briefly, but in sufficient detail to preserve the characteristics of the symptomatology.



## CIRCULAR TYPES.

CASE 1.—Gottgetren. Male, aged 10. Heredity untainted. At 7 concussion of the brain, followed by failure of memory. Two days later unruly and restless, followed by a period of depression. Sent to an asylum. Hallucinations of voices in his abdomen, also of sight. Fears he will be killed and believes that a patient removed one of his ribs. Recovery in about one year.

CASE 2.—Von Brero. Male, aged 13. Family history of insanity. Unusual behavior attributed to naughtiness first, then suddenly developed delusions that the neighbors were plotting against him and heard voices from the next house conspiring against him. He was going to be murdered. Became restless, profane and threatening, at times depressed. Sleep restless and fitful. Refused solid foods and wet his bed. Symptoms associated with movements like oscillations more or less regular and choreiform in type. A period of exaltation was followed by a period of depression. Excited periods were characterized by maniacal conditions, the depressed periods by stupor, confusion, hallucinations and atony. Choreiform movements were present only during stuporous periods and were intentional. The periods varied from six to twenty days and followed close upon each other. Cured in about one year.

CASE 3.—Zimmer. Female, aged 9. Insane history. At the age of 8 suddenly would whirl companions around in a dance and would leave the class-room for no reason. Said she had been accused of stealing; was infested with vermin; was unhappy and threatened suicide. Was admitted to Mendel's Clinic, where she showed symptoms of manic excitement alternating with melancholic depression, with at times periods of comparatively normal conditions intervening. Results not stated.

CASE 4.—Hahn. Male, aged 10. Acute mania following a blow. No heredity. Intelligent before. Was struck by a cane on the left frontal and parietal region. This was followed by pains in the head and a sensation as though the head was soft at the point of blow. Five days later he was listless and apathetic. Sensations of terror around the heart and hallucinations of sight and sound developed. There was self-reproach delirium, accompanied by right facial twitching, rhythmic opening and closing of the mouth. There was a manic condition, followed by depression. The next day the condition was aggravated by irritability. Struck his mother and barked like a dog. There was verbigeration. Cured in three months.

CASE 5.—Wells. Female, aged 15. Negative heredity. At 14, after hard work at school to secure a prize, developed melancholic depression; insisted upon having her dresses very short and ate little so as to retard development. Demanded a tight corset to compress her chest and thus prevent normal development. If opposed became violent. In another month chorea symptoms intervened for two weeks, but returned again in two months with other symptoms becoming more pronounced. She screamed violently and talked incoherently; exhibited hallucinations of sight; refused food, and reproached her mother for leaving her. Could not sleep. Seven months

any source of reflex irritation in her nose and her vision was properly corrected.

She was clever at school and was normal, cheerful and good-natured.

In 1898, when she was 12 years old, while listening to instruction for her first communion she became very much depressed. She was very unhappy; felt that she had been guilty of committing many sins. Every act she interpreted as a sin. The depression was very much worse upon awakening. She was much absorbed by these depressive thoughts and did not recognize at the time that they were delusions. These symptoms lasted about a month, when the depression cleared up entirely. Incidentally the epileptic attacks diminished gradually in frequency and in 1904 ceased entirely.

Morris F., aged 13, the second case of melancholia, applied to the Polyclinic Hospital February 16, 1914. Family history was negative. There had been no history of insanity or epilepsy or other nervous diseases. The patient had had measles, but had otherwise enjoyed good health. He was clever in his school work, having reached the Fifth Grade A.

His present illness began in June, 1913, when he was struck by a baseball over the left eye. He was not rendered unconscious and was able to leave the hospital in a day. He has been nervous and restless ever since. His father stated that he acted in a stupid manner; he slept very poorly; complained of headache, especially at night, and in the three weeks prior to his going to the hospital had dizzy spells. Two months ago he fell unconscious to the floor, but did not have any convulsion. Recently he became very much depressed, refused his food and did not play with his companions, sitting around the house all day long, voluntarily making no remark to anyone.

At the time of the examination he was apparently very much depressed. His face was expressionless, his eyes were fixed in one direction and he could be aroused from his reverie only with great difficulty. He denied hallucinations of hearing. He did not know why he was depressed or what he was worrying about. He mumbled his replies to the questions of the examiner, blinking his eyes and giving indefinite replies. He never smiled and said that he felt sad and was sick at heart. At times he would walk constantly around the house without any object. This patient is still under observation, having improved very considerably, though he had not entirely recovered when seen last.

In a thorough search of the literature available I collected forty-four cases which seemed to have been recorded sufficiently carefully to warrant quoting. These cases can be divided into the manic-depressive type, including the circular, manic, and melancholic types, post-infectious psychoses, and one case interesting as illustrating a precox type of psychosis which is not readily classed. These cases have been described briefly, but in sufficient detail to preserve the characteristics of the symptomatology.

## CIRCULAR TYPES.

CASE 1.—Gottgetren. Male, aged 10. Heredity untainted. At 7 concussion of the brain, followed by failure of memory. Two days later unruly and restless, followed by a period of depression. Sent to an asylum. Hallucinations of voices in his abdomen, also of sight. Fears he will be killed and believes that a patient removed one of his ribs. Recovery in about one year.

CASE 2.—Von Brero. Male, aged 13. Family history of insanity. Unusual behavior attributed to naughtiness first, then suddenly developed delusions that the neighbors were plotting against him and heard voices from the next house conspiring against him. He was going to be murdered. Became restless, profane and threatening, at times depressed. Sleep restless and fitful. Refused solid foods and wet his bed. Symptoms associated with movements like oscillations more or less regular and choreiform in type. A period of exaltation was followed by a period of depression. Excited periods were characterized by maniacal conditions, the depressed periods by stupor, confusion, hallucinations and atony. Choreiform movements were present only during stuporous periods and were intentional. The periods varied from six to twenty days and followed close upon each other. Cured in about one year.

CASE 3.—Zimmer. Female, aged 9. Insane history. At the age of 8 suddenly would whirl companions around in a dance and would leave the class-room for no reason. Said she had been accused of stealing; was infested with vermin; was unhappy and threatened suicide. Was admitted to Mendel's Clinic, where she showed symptoms of manic excitement alternating with melancholic depression, with at times periods of comparatively normal conditions intervening. Results not stated.

CASE 4.—Hahn. Male, aged 10. Acute mania following a blow. No heredity. Intelligent before. Was struck by a cane on the left frontal and parietal region. This was followed by pains in the head and a sensation as though the head was soft at the point of blow. Five days later he was listless and apathetic. Sensations of terror around the heart and hallucinations of sight and sound developed. There was self-reproach delirium, accompanied by right facial twitching, rhythmic opening and closing of the mouth. There was a manic condition, followed by depression. The next day the condition was aggravated by irritability. Struck his mother and barked like a dog. There was verbigeration. Cured in three months.

CASE 5.—Wells. Female, aged 15. Negative heredity. At 14, after hard work at school to secure a prize, developed melancholic depression; insisted upon having her dresses very short and ate little so as to retard development. Demanded a tight corset to compress her chest and thus prevent normal development. If opposed became violent. In another month chorea symptoms intervened for two weeks, but returned again in two months with other symptoms becoming more pronounced. She screamed violently and talked incoherently; exhibited hallucinations of sight; refused food, and reproached her mother for leaving her. Could not sleep. Seven months

later she improved very much, but the symptoms returned in a month and she had to be removed from her home on account of being so noisy. She improved at the end of a year and eight months from the beginning of the disease and remained well.

CASE 6.—Tremoth. Male, aged 14. Heredity positive. Normal before onset. Became reserved and apathetic and listless, refused to take food and lay perfectly listless; incoherent and mumbling. Later state of fear and tremor; loud inarticulate words for a day or two. Later he suddenly became lively, unruly, destructive, but still mute for an hour. This was repeated on two other occasions. Recovered in three months.

CASE 7.—Tremoth. Male, aged 13. Mentally deranged. At the age of 12 began to have hallucinations and feared that someone was going to hurt him. These attacks of fear recurred every eight weeks. During the interim periods of unwonted hilarity and sprightliness. Cured at the end of a year.

CASE 8.—Lehr. Male, aged 9. No heredity. Under strict supervision of studies there developed fear and melancholic depression, followed by states of excitement and rage, stubbornness and unruliness. Recovered completely.

CASE 9.—Esquirol. Female, aged 8. Normal intelligence. Sustained a severe shock during the storming of Paris and showed signs of mental disease. There were states of somnolence, alternating with unruliness, noisiness and vulgarity.

CASE 10.—Esquirol. Male, aged 15. Illegitimate. Heredity taint. Sudden violence and incessant talking, alternated with periods of misery and depression. Improvement finally set in.

CASE 11.—Ray. Female, aged 14. First symptom loss of memory, then became violent and uncertain, refusing food; dirty and untidy. She moaned piteously. Later she became self-absorbed so that she failed to understand the simplest orders. She recovered.

CASE 12.—Nasse. Female, aged 15. Poor heredity. Began with melancholia at 14 and developed into periodic mania.

#### MANIC TYPES.

CASE 13.—Fletcher. Female, aged 5. After being threatened with being shut up in a closet and having heard tales of dark places where witches live and the like, was very much frightened and became pale and cold and for months thereafter raved and muttered about hobgoblins. When she recovered she was very much changed.

CASE 14.—Fletcher. Male, aged 15. Intelligent. Suddenly became profane and began to talk incessantly of himself; smoked cigarettes excessively and boasted of his capacity for strong drink and his success at cards and his escape from hairbreadth adventures. He believed that he was a detective. Recovered.

CASE 15.—Fletcher. Male, aged 10. Neurotic parents. Imagined himself an Indian and was uncontrollable, attacking his parents and shooting at

them with arrows. He wandered in the woods at night. He had attacks of convulsions which lacked the elements of epilepsy. Recovered.

CASE 16.—Holmes. Male, aged 13. Heredity negative. After a quarrel with his sister became very much excited and threatened violence against his mother and himself. At first he was mischievous, singing and whistling and talking incoherently. He regained his normal tone in ten days.

CASE 17.—Holmes. Male, aged 10. Average intelligence. After a fall, in which he fractured his humerus, seemed to be too happy. He was irrational and showed flurries of excitement and talked excessively about his accident. Normal in four weeks.

CASE 18.—Holmes. Female, aged 7. Average intelligence. Negative history. Suddenly began to talk excessively, rhymed and commented constantly on what went on around her. Her attention was gained with difficulty. She exhibited much motor excitement. Cured in six weeks.

CASE 19.—Liebers. Male, aged 5. No heredity. Following diphtheria at 4 was unable to talk or walk for six weeks and then became restless and destructive, refusing to eat or sleep. Stands up in bed and laughs gleefully and throws his arm affectionately around the doctor's neck and then throws himself back in bed again. Violent motions and grimaces in a perfectly tireless manner. Attention could not be gained without great effort. Duration one year, followed by cure, although feeble-minded.

CASE 20.—Schuller. Male, aged 6½ years. Well until one year previously, then an indefinite fever lasting about a week. Then began to act strangely; talked to himself for hours; refused to eat; lost control of his functions; could not talk; countenance smiling and walked with a peculiar hop. Some improvement.

CASE 21.—Engelhorn. Male, aged 11. Became violent and excited after an explosion in the garden of his home which caused the death of his brother. Symptoms lasted only a short time.

CASE 22.—Steiner. Male, aged 6. Showed periodic mania. There were periodic paroxysms of rage requiring strait-jackets and opiates. Result idiocy.

CASE 23.—Holmes. Male, aged 13. Heredity negative. After a quarrel with his sister became very much excited and threatened violence against his mother and himself. At first he was mischievous and singing and whistling and talking incoherently. He regained his normal tone in ten days.

#### DEPRESSIVE TYPES.

CASE 24.—Noyes. Male, aged 13. Heredity negative. Mental depression and crying spells. He said his fellow pupils did not like him and accused him of onanism. Some choreiform movements, but not chorea. Cured in two years.

CASE 25.—Noyes. Male, aged 13. History negative. Restless, crying spells, anemia and insomnia; talked obscenely to his mother. Complained of being abused by the other boys. Mental depression, everybody was making fun of him. He became hyperesthetic and complained of globus hy-



stericus. Some choreiform movements, but not chorea. Cured in six months.

CASE 26.—Beach. Male, aged 14. After attending a funeral of a friend who had committed suicide said, "I must kill myself, too," and seeing the cord at the place of the suicide proceeded to take his own life.

CASE 27.—Ireland. Female, aged 11. Melancholia. She had nose bleeds appearing with attacks of loud wailings. Heredity and alcoholism were emphasized as predisposing causes. Recovered in a year.

CASE 28.—Fletcher. Female, aged 13. After the death of her mother was sent away to school. On her return from vacation she became melancholy and refused to work, spending most of the time in a semi-conscious condition in bed. She told a neighbor that she was pregnant and that her father had seduced her. Father later became insane, but the patient apparently recovered at the end of four years.

CASE 29.—Tremoth. Male, aged 16. Uncle insane. Intelligent and normal previously. Suddenly grew quiet and avoided seeing people and was hallucinatory. He lay with his eyes closed, refusing food, and was full of unaccountable terrors. Cured in six months and remained normal.

CASE 30.—Savage. Female, aged 11. Intelligent. Became very religious at 10, seeing angels and communing with them. Began to menstruate then. Then lost faith in religion and attempted to kill herself. Intense likes and dislikes, with precocious tendency towards the other sex.

CASE 31.—Gregory. Female, aged 10. Negative heredity. Normal child. After a disappointment became sad and quiet; complained of headache and refused to eat. Upon examination she was profoundly depressed; her head and body bent forward. There was no spontaneous speech. She complained of headache and pain in the stomach. She was oriented and her memory was good. No hallucinations or delusions. She improved at the end of three or four weeks, but the depression returned in a few days. She recovered.

#### POST-INFECTIOUS PSYCHOSES.

CASE 32.—Kuhn. Female, aged 6. Exhibited acute stuporous dementia with epileptic attacks and chorea following diphtheria.

CASE 33.—Mischede. A child of 5 years and 9 months following whoop-cough developed hallucinations of sight and hearing, which were preceded by sudden feelings of heat and cold.

CASE 34.—Welt. Female, aged 12½. Family history good, except for neuroses. Two weeks after convalescence from typhoid became depressed with insomnia and anorexia. She could not rid herself of the idea that she should kill her mother. Recovered in a few weeks.

CASE 35.—Welt. Male, aged 5. Good health. After an attack of scarlet fever became restless and irritable; did not recognize his mother; became terror stricken; wanted to rush out of the house because the house was on fire. Staring countenance. Recovered in forty-eight hours.

CASE 36.—Gerlach. Female, aged 10. Insane history. Normal child. October scarlet fever; January nephritis. March 10 suddenly had spasms,

which lasted forty-eight hours. Aphasia ensued. She became irritable, quarrelsome and destructive. Dirty in her habits and crawled around on her hands and feet and mumbled.

This condition lasted for two months. Her speech had returned, but was monotonous, alternating with clear, lively and rapid speech. She had hallucinations of sight and hearing and she could not feed herself. Sensibility seemed to be lost except on the soles of the feet. There was an attack of angina later with a rapid pulse. Great motor weakness ensued. Four months after the onset there was contracture at the left knee, with increased reflexes. She began to improve gradually and was cured at the end of nine months. She described her symptoms afterwards as follows: She had a feeling of terror; someone was about to kill her. She saw black figures on the ceiling and members of her family standing about her bed scolding her. She did not speak, because she was afraid. Gerlach believed that the symptoms were all the result of uremia, which was favored by heredity.

CASE 37.—Joachim. Male, aged  $4\frac{1}{2}$ . Heredity stigma. During convalescence from scarlet fever, complicated by abscesses in the throat, suddenly developed raging frenzy and hallucinations, with symptoms of collapse. Refused food; was destructive and passed her urine and feces involuntarily. Symptoms disappeared in forty-eight hours.

CASE 38.—Hume. Male, aged  $2\frac{1}{2}$ . Intelligent, neat and orderly, but after catching a cold during an attack of malarial fever became restless, biting paper and rubbish and was destructive. His speech, previously plain, became indistinct. Cured in two weeks.

CASE 39.—Jensen. Male, aged 9. Negative history. During convalescence from typhoid fever suddenly became very talkative. There was emotional exaltation and motor agitation, anger or tears over trifles and then delusions of grandeur. Recovered in about four weeks.

CASE 40.—Jordan. Male, aged 7. During convalescence of typhoid fever four weeks after the temperature had become normal became suddenly violent and had hallucinations of sight. Frogs crawled about; he had only one arm, was incoherent; lost control of urine and feces; was sullen and suspicious. Recovered in four weeks; no rise of temperature during the attack.

CASE 41.—Feith. Male, aged 5. Negative heredity. Typhoid fever, followed by pronounced motor disturbance, aphasia combined with fear and melancholic depression lasting three weeks. Sudden return of speech and the child became lively and unruly and for two weeks talked incessantly and was in a continual state of exaltation. Recovered.

CASE 42.—Welt. Male, aged 10. Negative heredity. Intelligent and studious. After an attack of diphtheritic sore throat and albuminuria he began to be restless, talkative and impudent. This was followed by paroxysms of rage, destructiveness and brutality. Bit and choked his mother; tore the clothing from her body. These paroxysms were sometimes preceded by periods of stupor, staring countenance, bodily rigidity. Hallucinations were rare and were those of sight. Complete recovery in about five months.

CASE 43.—Nierier. Male, aged 13. Periodic frenzy after typhoid fever, then alternating depression and exaltation.

CASE 44.—(This case suggests an early dementia precox.) Wells. Male, aged 14. Somewhat below average intelligence. Left school at 12 and went to work as a cash boy; at which age he developed persecutory hallucinations; wandered the streets for three days before apprehended and could give no account of his whereabouts during this time. Hereafter returned to school, but one day did not come home and was found a week later in a vacant lot ragged and terrified. Was returned to school, where he remained for six months, making fair progress, although showed some evidence of being suspicious of those about him.

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## EPILEPTIC DEMENTIA.\*

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Epilepsy may be associated with all forms of mental disturbances. It is met with in the course of functional or organic psychoses. During the evolution of symptoms of paresis, paranoia, dementia præcox, manic-depressive insanity epileptic seizures may occur. A temporary or transient mental manifestation, such as hallucination or a stuporous or delirious state, may precede or follow epileptic seizures. It may also take the place of an epileptic attack and become an equivalent of the latter.

We will not be concerned with all these occurrences, but exclusively with the close connection between epilepsy and quantitative diminution of intellect, viz., dementia. Quantitative diminution of intellect, viz., dementia, may also be met with alongside of epilepsy. There we find all varieties. The dementia may have existed before the epilepsy, or both conditions developed simultaneously after a cranial traumatism, or the dementia and epilepsy are both the result of some cerebral lesion, or finally the dementia developed some time later after the onset of epilepsy.

The first variety belongs to the class of idiots and imbeciles. There are usually material causes for both conditions such as encephalitis, hydrocephalus, meningitis, cerebral hypoplasia or hyperplasia and other malformations of the central nervous system. In these cases the mental development had been arrested in early childhood. The individuals are either idiots or imbeciles and they are commonly affected with epileptic attacks. In such cases epilepsy merely accompanies the intellectual deficit, but there is not necessarily a direct relation between the two as cause and effect. The cerebral lesion or malformation interfered with the development of the highest centers, hence the mental arrest, and at the same time served as an irritating factor to the cerebral tissue, hence the epilepsy.

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In the second variety, in which there is a history of trauma, epilepsy may appear months or years after the cranial traumatism. The cerebral lesion produced by the latter determines the epilepsy. The dementia may appear simultaneously with the epilepsy and in some cases the dementia precedes the epilepsy. Here again there may be no direct relationship between the two conditions. The trauma and the material lesion of the brain are the individual causes of both.

Not only in cases of traumatism but also in lesions of the brain from other causes, in chronic meningo-encephalitis for example, epilepsy and dementia may accompany each other without direct relationship between the two.

Let us consider now the cases in which dementia is the direct result of epilepsy. They constitute genuine cases of epileptic dementia. This study embraces eight patients who have been kept under observation for a period of twelve years and in which the dementia developed subsequently to a more or less prolonged period of epileptic seizures. The patients of these series presented the following peculiarities as to the period of life at which the disturbances made their appearance.

Five individuals developed epilepsy at the age of puberty, one at 27 years of age, one at 37 and one at 62. The last two patients began to show symptoms of dementia early in the course of their epileptic seizures. In the other six patients signs of mental enfeeblement were observed three or five years after the onset of epilepsy. With the exception of the woman aged 62, who began to show symptoms of dementia four months after the onset of epilepsy, in all the other patients the mental enfeeblement made its first appearance when the epileptic seizures became very frequent. As to the ages at which both morbid conditions appeared, they are seen in the following table:

Patients.	Age of the Onset of Epilepsy.	Age of the Onset of Dementia.
1 (girl) .....	15	17
2 (girl) .....	15	17
3 (girl) .....	16 years 5 months	20
4 (girl) .....	16 years 3 months	20
5 (boy) .....	16	19
6 (man) .....	27	31
7 (man) .....	37	40
8 (woman) .....	62	62 years 4 months

It was interesting to observe that the adolescent group of patients reminded in some respects the clinical picture of dementia præcox. We find here apathy, indolence, some vague delusional ideas and in some cases catatonic attitudes. These are the manifestations in which resemblance existed. But it is also interesting to note that while in dementia præcox the anterior intellectual acquisitions disappear as a rule quite rapidly, in cases of my series in which the dementia was associated with and dependent on epilepsy the diminution of memory for already acquired knowledge was very gradual.

Constitutional diseases, such as tuberculosis, diabetes, etc., were all observed in the histories of the entire series of my patients. Syphilis could equally be excluded as a Wassermann test on the blood serum, and cerebrospinal fluid as well as Noguchi test proved negative in every one of the patients.

Alcoholism was present in the man aged 37. He commenced to drink at the age of 30. Quite considerable quantities of alcohol were consumed by him. However, he never presented alcoholic delirium or any other mental disturbances. His epileptic condition developed at the age of 37 following an alcoholic debauch. At first the seizures were frequent, but at the end of six months they became rare. At 39 the seizures became again frequent and occurred once about every week or every two weeks. It is interesting to note that from the time epilepsy developed the patient did not resume the use of alcohol except on two occasions at a year's interval, when he drank very moderately (2 or 3 drinks of whiskey in one week). At the age of 40 symptoms of mental enfeeblement commenced to become manifest. Memory, attention, power of concentration, initiative and finally judgment—all these faculties underwent gradual diminution. The interesting feature about this case is that alcohol cannot be directly incriminated as the cause of the mental disturbances. It is the epilepsy which is closely and chronologically associated with the dementia. Nevertheless, there is a previous history of alcoholism which may have prepared the individual to the mental disorder and the epileptic seizures served as a reinforcement.

The woman of 62 presented physical symptoms of marked senility. Arcus senilis, arteriosclerosis of the most pronounced type, high blood pressure, almost musical accentuation of the

second aortic sound, presbyopia, are the manifestations observed in this individual. Without an apparent exciting cause she developed epileptic seizures of the usual type. At first rare, they soon became frequent so that during the third month they occurred twice a week. Four months later the patient began to show signs of pathological mental senility. Actual diminution of sustained attention, weakness of memory, a certain childishness in acting and feeling—soon became manifest. In this case the oncoming physiological senility undoubtedly had a predisposing influence on the development of dementia which made its appearance *apropos* of repeated cortical irritation caused by the epilepsy.

It is evident that in the last two cases there are elements that may have played a certain rôle in the causation of the dementia and the epilepsy could be considered both as an exciting and direct factor. Such unfavorable elements are entirely absent in the six other patients. Here the mental enfeeblement appears to be the direct consequence of the epilepsy. It is interesting to note that the younger the individual, the earlier signs of mental disturbance would make their appearance. It is also important to know that in all the six patients, while there are no personal medical antecedents of any moment, nevertheless their family histories present certain features which point to a neuropathic heredity. In the two girls of 15 there is a history of alcoholism in the parents. The girl of 15 years 5 months had a sister who was confined to an asylum. The families of the other four patients presented some mental abnormalities which it was very difficult to determine with accuracy. The younger individuals, including the man of 27, grew up to all appearances normally, when at their respective ages epilepsy developed. The two girls of 15 had at first attacks of petit mal exclusively, but soon major attacks supervened. The other patients commenced their epilepsy with seizures of grand mal. All the six young patients as well as the last two older individuals presented major seizures exclusively at the time the symptoms of mental enfeeblement began to appear. As to the character of the epileptic attacks, they were all of the generalized type. In none of them unilaterality of the convulsions was observed.

Let us consider now the chief features of the dementia as they appeared in my eight patients. The clinical pictures differed in the younger and in the older individuals. In the first group, as it

was intimated above, the resemblance to dementia præcox is striking. The absence of any mental abnormalities in the lives of this group of patients prior to the onset of epilepsy excludes at once any possible influence on the mental enfeeblement other than the epilepsy. It seems there was a direct relationship between the two disorders.

The similarity to dementia præcox was more than ordinary. We find here involvement of the affectivity, of judgment, diminution of memory for recent and old events, feebleness of comprehension and concentration. In every one of them could be observed delusional conceptions of a transitory character; also hallucinatory images. In spite of this striking similarity between these two affections, there was one feature which remained so conspicuous and so persistent that it appealed as a diagnostic indication in differentiation of both diseases. It consisted of the preservation to a large degree and for a long time of the previous intellectual accomplishments acquired prior to the onset of epilepsy. The youngest of my patients was 15 years of age; the knowledge obtained up to that age was quite considerable. In spite of the gradual oncoming of other symptoms of dementia and in spite of the inability of acquiring and assimilating new facts, the memory for already acquired data remained intact for a long time. Their diminution and disappearance was exceedingly gradual and insidious—a condition which is not observed in dementia præcox. In the latter affection intellectual acquisitions usually disappear quite rapidly.

This special feature which appeals to me as being characteristic was particularly noticeable in the men of 27 and of 37. Here the retention of the anteriorly acquired knowledge persisted with greater integrity and for a more prolonged period than in the much younger individuals. It must be mentioned, however, that in all those individuals the preservation of facts concerning previously acquired knowledge was more in a quantitative than in a qualitative sense of the term. When the faculties of judgment, of discrimination, began to fail, the power of criticism commenced to suffer. The patients, while able to recall important data, showed at the same time lack of their full appreciation and weakness in criticising them. This condition existed throughout the entire period of the gradual decrease in the mental power.



The two patients of 27 and 37 years of age presented in addition to the last-mentioned characteristics also some features of paresis. The speech, while not entirely typical of the latter, nevertheless reminded at times of a paretic speech. The facies, the attitude, the occasional syllabic manner of speaking, the fine tremor of the hands, together with the frequent attacks of epilepsy—all rendered the dementia of my two patients strongly similar to paretic dementia. However, an analysis of the cerebrospinal fluid showed absence of lymphocytosis and negative both Wassermann and Noguchi reactions, all facts which exclude at once the diagnosis of paresis.

The last patient of my entire series, the woman of 62, presented as a special feature symptoms resembling those of senile dementia. In fact, at first glance no other condition could be thought of, and were it not for the underlying characteristics of the so-called epileptic character of the individual, the patient could have been considered as suffering from pathological senility. But in view of the fact that the former normal mentality became abnormal shortly after the onset of epilepsy, and in view of the above-mentioned characteristics, the dementia must be directly associated with the epilepsy.

Apart from the special features characteristic of the separated groups of my cases as considered above and in spite of these fundamental differences, a particular set of characteristics could be found common to all the patients of my series. They were: undue irritability, leading at times to brutal acts; outbursts of passion, anger and impulsiveness; hostility; distrust; loss of affective faculties; perversion of moral sense; egotism; extreme sensitiveness; mysticism. These characteristics constitute a very important set of phenomena, which were so uniformly observed in all my cases that they could be considered typical of epilepsy. They served as a diagnostic guide in the process of analyzing the mental attitude of my patients and in determining the direct relationship between the psychic manifestations and the epilepsy itself. They helped to establish the fact that while some symptoms of dementia præcox, of paretic dementia and of senile dementia were present, nevertheless, there was merely a resemblance to these psychoses. The above-mentioned characteristics carefully studied and observed throughout a long period of time enabled me to make a

sufficiently distinct differentiation between those maladies and the mentality of my patients.

The conclusions which the above observations permit to draw are as follows:

1. Normal intellectual faculties may coexist with epilepsy, provided the latter is not frequent.
2. Should epilepsy become frequent, it eventually exercises a destructive influence on the mind. The latter consists of retrogression of intellectual power, viz., a gradual, progressive feebleness of intellect, leading to dementia.
3. Epileptic dementia may develop at any age.
4. Should epilepsy appear at the age of adolescence, the dementia assumes the clinical picture of dementia præcox.
5. When epilepsy develops in an adult, the dementia may resemble that of paresis.
6. If an elderly person suffers from epileptic seizures, his mental condition may simulate senile dementia.
7. A thorough analysis of all such cases will reveal the fact that the resemblance to the above diseases is not a profound one; the individual characteristics of epileptics which are invariably present will soon enable one to ascertain the true nature of epileptic dementia.
8. The majority of epileptic demented present family antecedents of a neuropathic nature.



## SOME NOTES ON EXPERT TESTIMONY BY ALIENISTS AND NEUROLOGISTS.\*

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I have been prompted to write this paper principally because it concerns a civil case of much prominence recently heard in the Montreal courts. Dr. T. J. W. Burgess and the writer were summoned, both being witnesses as to fact; the plaintiff's father at the time was a patient at Verdun Hospital.

Such a paper, it must be confessed, brought before this Society, smacks strongly of "Carrying coals to Newcastle," when one considers the many excellent monographs on this subject presented to you in the past few years. My apology must be that a dart, though feebly delivered at an evil, may strike its mark. The writer assumes there is no disagreement among the members of the profession with the assertion that there is room for improvement in the methods allowed and practised, on the one hand, regarding the securing and hearing of expert testimony, and the giving of it, on the other, in the courts of the United States and Canada. This applies particularly to civil cases which hinge upon proof or non-proof of insanity, an example of which it is hoped to demonstrate in this paper; certainly, the contradictory character of the evidence submitted by the various experts in the case here dealt with, calls for some remedy.

Thirteen physicians gave evidence in the case herewith sketched, eleven of whom have made a special study of psychiatry or neurology. The review of their statements, under oath, which forms the major part of this paper, furnishes one of the most instructive examples as to why expert testimony, by alienists especially, in Canada and the United States is to-day liable to be received with a damning smile of tolerance that can well be imagined. The spectacle of reputable medical men (for be it understood that all concerned were, and are highly so) making depositions so bluntly opposed to each other, can but cast a shadow on the physician's

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reliability or knowledge, or both, when he is in the witness box—whatever he may be when outside of it.

The fact might as well be squarely faced that a physician is but human, and notwithstanding the high ethical and professional standards to which he is bound, and in justice be it said, to which he faithfully adheres, the influences of partizanship, personal feelings, and the desire that his side shall win in the courtroom, have, in degree, an effect upon his testimony, as upon that of the non-professional and inexpert witness. A prominent and most able judge of Toronto, Can., not so long ago remarked to the writer that even in his official capacity, when hearing evidence, he felt that subconsciously he gave heed to the very expression of a witness, his manner and his method of answering questions under cross-examination, and that while all these points were distinctly not pertinent to the evidence, they, to a limited extent, tended to bias his belief as to the value of the testimony adduced, either favorably or unfavorably. If a man, with a wealth of experience on the bench, will honestly make such a statement, it would be surprising indeed if the quasi-expert testator could prevent himself from showing some trace of partiality, although he might intend and desire to preserve the aloofness and detachment that one totally disinterested as to the issue in a given case should manifest.

The spirit in which this paper is written is not one of arraignment; it is intended to point out, by a practical example, that the matter of experts and their selection, etc., deserves the most serious deliberation by legislator and physician alike. It is hardly possible in a paper of this scope to attempt suggestions as to how this may be best done. Some practical ideas by an eminent English judge, which bear upon the point of how more unanimity of opinion may be arrived at among medical witnesses, is worth reading, and I quote it with pleasure. In "A History of the Criminal Law of England," by Sir James Fitzjames Stephen, K. C. (Edition 1883, page 575), he says:

It is impossible to say what an expert is to be, if he is not to be a witness like other witnesses. If he is to decide upon medical or other scientific questions connected with the case so as to bind either the judge or the jury, the inevitable result is a divided responsibility which would destroy the whole value of the trial. If the expert is to tell the jury what is the law—say, about madness—he supersedes the judge. If he is to decide whether, in fact, the prisoner is mad, he supersedes the jury. If



he is only to advise the court, is he or is he not to do so publicly, and to be liable to cross-examination? If yes, he is a witness like any other; if no, he will be placed in a position opposed to all principle. The judge and the jury alike are, and ought to be, instructed only by witnesses publicly testifying in open court on oath.

It never would be, and never ought to be, endured for a moment that a judge should have irresponsible advisers protected against cross-examination.

And proceeding, he says:

The truth is, that the demand for experts is simply a protest made by medical men against cross-examination. They are not accustomed to it and they do not like it, but I should say that no class of witnesses ought to be so carefully watched and so strictly cross-examined.

There is one way in which medical men may altogether avoid the inconveniences of which they complain, and that is by knowing their business and giving their testimony with absolute candor and frankness. There have been, no doubt, and there still occasionally are, scenes between medical witnesses and the counsel who cross-examine them which are not creditable, but the reason is that medical witnesses in such cases are not really witnesses, but counsel in disguise, who have come to support the side by which they are called. The practice is, happily, rarer than it used to be, but when it occurs it can be met and exposed only by the most searching, and no doubt unpleasant, questioning. By proper means it may be wholly avoided. If medical men laid down for themselves a positive rule that they would not give evidence, unless before doing so they met in consultation the medical men to be called on the other side and exchanged their views fully, so that the medical witnesses on the one side might know what was to be said by the medical witnesses on the other, they would be able to give a full and impartial account of the case, which would not provoke cross-examination. For many years this course has been invariably pursued by all the most eminent physicians and surgeons in Leeds, and the result is that, in trials at Leeds (where actions for injuries in railway accidents and the like are very common), the medical witnesses are hardly ever cross-examined at all, and it is by no means uncommon for them to be called on one side only. Such a practice, of course, implies a high standard of honor and professional knowledge on the part of the witnesses employed to give evidence; but this is a matter for medical men. If they steadily refuse to act as counsel, and insist on knowing what is to be said on both sides before they testify, they need not fear cross-examination.

I would also refer to the law in France regarding expert witnesses, from which we in Canada, as well as the United States, might glean much of profit to improve faults common to both countries.

In "Précis de Medecine Legale," by L. Thoinot, professor of legal medicine of the Faculty of Medicine of Paris (Edition 1913, Vol. I, pages 36-37), we learn that the nomination of medical experts in France is governed by a decree of November, 1892, Art. XIV, which states that "The duties of medical experts can only be filled by medical men holding French degrees." As to the appointment of medical experts by a decree of November, 1893, Art. I, we find the following: "At the commencement of each judicial year and in the month which follows its opening, the Courts of Appeal in Chambers, the Attorney-General being present, will designate upon their lists the doctors in medicine on whom they will confer the title of expert before the courts." Article II, amended by the decree of April, 1906, states: "The nominees of the court must be graduates of a French medical school residing within the jurisdiction of the court. They must have five years practice in their profession or have a diploma from the University of Paris bearing the special qualification of Médecine légale et Psychiatrie, or a similar diploma created by the other French universities."

The French Republic, certainly not a backward nation in legal matters, deems medico-legal experts—and it follows their testimony—of sufficiently grave importance to officially designate who shall be deemed worthy of this title. These experts are appointed yearly, by one of the most powerful judicial bodies of France, and are invariably men fully qualified; they must reside in the judicial district in which the trial takes place, none being called from outside the "arrondissement" of the tribunal, *i. e.*, the district over which a particular court presides, and a special diploma is exacted in lieu of five years' practice. Such a system unquestionably makes for a high standard in the personnel of the medical expert, and surely it cannot but place his testimony on a plane not easily assailed by even the most astute attorney, as well as armor it against cavil by the laity.

Now as to the case at issue: First, the salient points regarding certain physical features and the character of the psychosis from which the patient suffered may well be presented.

G. M., Montreal. Previous History: Admitted July 15, 1912, to Verdun Hospital, act. 66; first attack manifested itself November, 1911, in insomnia, irregularities in finance; at present shows visual hallucinations;

suicidal propensities; capricious appetite; insomnia; heavy drinker for thirty years; progressive muscular atrophy evident in wasting muscles of right thenar eminence; has lost weight; has arteriosclerosis; emotionally depressed and states he contemplated suicide.

On Admission: Depressed; speaks of suicide and claims to have attempted it; delusion that detectives are continually on his track; says he lost several hundred thousand dollars in poor business deals and that he is financially ruined; worrying greatly over his son, who is now seriously ill with typhoid; sleepless.

*Abstract of Physical Examination.*—Pulse 68. Circulatory System: Arteriosclerosis of radial and temporal arteries evident; blood pressure 168, pulse pressure 65. Kidneys: Examination of urine; nothing abnormal. Syncopal attacks several (five or six).

*Diagnosis.*—Melancholia of involution type.

The litigation in the case centers about these facts. The patient, G. M., was certified as insane in July, 1912, and committed to Verdun Hospital in that month. In the months of February and March, 1912, he had made over certain bonds and securities, worth some \$70,000, to his nephew, W. M. The patient's son, R. M., claimed that this collateral was given to the nephew without adequate consideration of any kind and that his father's mind was affected when he made the transaction. He, R. M., therefore brought suit against W. M. to recover the said securities, etc. The question to be decided resolved itself into, whether when G. M. transferred this valuable collateral to his nephew he was in a fit mental state to do so or not. Both sides called a number of expert witnesses, and the vital portion of their testimony is appended in juxtaposition. Were any one of you who read this article called upon to be the judge, from the learned expert testimony presented, what would your decision be? You are not required to give your opinion on the value of expert evidence in the connection; it were better left to the imagination. I have totally disregarded in this paper what has been said by the lay witnesses. It has no particular place, as it is quite non-technical. Suffice it to say that it brings much to prove the man was absolutely insane and perfectly sane when he consummated the business deal out of which the suit arose.

A brief hearing of the depositions of Dr. S. and Dr. T., the physicians who saw the patient in January and March, 1912, should first be considered. Neither affected alienism or neurology, both being general practitioners. Their declarations seem of especial

value, owing to the symptoms which both observed, and the early dates, viz., January and March, 1912, on which they saw the patient.

Deposition of Dr. S., a witness produced and examined on behalf of the plaintiff in this matter.

Examined by H. J. E., counsel for plaintiff.

Q. Will you describe to us the condition in which you found the interdict, G. M., when you made your first examination in January, 1912?

A. A physical examination developed a condition of marked arteriosclerosis, or hardening of the arteries, enlargement of the heart, and wasting of the muscles of the hand. The nervous symptoms at the time were insomnia, great agitation and inability to fix his mind on what I talked to him about.

By the Court:

Q. Inability of concentration?

A. Yes. A "stary" look when you talked to him for a while, and you had to recall him to himself in order to get him to answer questions correctly. He also had an idea that he had ruined his boy.

Mr. E., continuing:

Q. When was this?

A. Early in January. I prescribed for him and the case went along. At times I heard from Mrs. M. that he was still walking the floor at night, and unable to sleep; that he was taciturn and would not talk. The next act of importance in his case was toward the end of February, when he had a revolver and thought he would do away with himself. Mrs. M. took the revolver from him.

Q. From your examination of the patient up till that time, and from your practically constant observation of the man, what is your opinion, as a medical man, as to his mental condition at that time?

A. I think he was mentally unfit at that time, and he was gradually getting worse.

Q. What condition did you find him in prior to the 5th of March, when you advised his incarceration in a sanitarium? Was he sane or insane?

A. Insane.

By the Court:

Q. And it goes without saying that he was insane in June when you came back?

A. Yes.

By Mr. E., continuing:

Q. In your opinion, what would be the effect of such a condition on his capability to conduct business?

A. I don't think he was fit, for the simple reason that he could not concentrate his mind on the business he had to perform.

Deposition of Dr. T., a witness produced and examined on behalf of the plaintiff in this matter.

Examined by Mr. S., counsel for plaintiff.

Q. Do you know G. M., now a patient in the Verdun Asylum?

A. Yes, I do.

Q. How long have you known him?

A. I have known him since the beginning of his son's illness. As far as I can remember, it may have been September or October, 1911. It was the beginning of the autumn of the year he was taken ill. As far as I can remember the first time I saw Mr. G. M. professionally was some time in the first two weeks in March. I could not be quite precise about the date.

Q. March, 1912?

A. Yes.

Q. Some time in the first two weeks of March?

A. Yes, as near as I can remember.

Q. What was his condition at that time?

A. When I saw him I remember it was in the late afternoon. It was pretty dark in the room where he was. I saw him sitting in a chair looking very downcast. I sat down and began to question him. I asked him what the matter was, and I told him that Mrs. M. was worried about him and had asked me to see him. At first I could not get any answers at all. He simply sat there in a gloomy condition, and I had the greatest difficulty in getting any information at all from him. I asked him whether he felt ill himself, that is, whether he was physically ill, and he said, "No." I then asked him what was the matter, and he said, I cannot give you his exact words, because I cannot pretend to do so at this distance, but he said, "It is terrible. I have ruined my family." That was really the first intimation I had that there was anything wrong with him.

Q. What idea did you form, if any, as to the character of the mental trouble from which he was suffering?

A. Well, I came, first of all, to the immediate conclusion that he was profoundly melancholic. Nobody could have seen that man in the attitude in which he was, and the way he answered questions, without coming to the conclusion that he was in the larger sense melancholic.

Q. Will you tell the court the opinion you formed as to whether he was rational or irrational?

A. I may say, whenever I spoke to him, asking him what he felt like, or how he was, he would always come back upon this same idea that he had ruined his family. That was the invariable topic which he drifted back to, and that without my suggestion. If you asked him what was the matter with him he would answer, "Oh, I have ruined my family. It is terrible." That was the usual way he spoke.

Q. Did you tender any advice to the family concerning him during that period?



A. I did, yes. The advice was that, in the first place, I considered Mr. G. M. was not in his right mind, and that it was a question as to what should be done with him.

None of the alienists or neurologists whose evidence is subsequently submitted examined the patient until after he had been committed to Verdun Hospital in July, 1912; they were unanimous in their opinion as to his condition when seen by them; they all state that the man was a case of involution melancholia beyond doubt. It is in their deductions as to how long the disease had existed *before* they saw the patient, that their amazing differences of interpretation are manifested.

EXTRACTS FROM EXPERT  
TESTIMONY, AND CROSS-  
EXAMINATION THEREON,  
AS GIVEN FOR PLAINTIFF.

DEPOSITION OF DR. U.

Examined by Mr. C. H. S., of  
counsel for plaintiff.

Q. Dr. U., you are Superintendent  
of the ——— Hospital for the  
Insane?

A. Yes.

Q. It is recognized by the Govern-  
ment of the Province of Quebec?

A. Oh, yes. As a matter of fact,  
I am appointed as superintendent by  
the government.

Q. How long have you occupied  
the position of superintendent of  
this institution?

A. Twenty-three years.

Q. Before that, were you in the  
same profession?

A. I have been forty years in the  
same profession.

Q. As a specialist in mental dis-  
eases?

A. Yes. Very nearly forty years.  
Practically forty years.

Q. Have you among the patients  
at the ——— Hospital for the In-  
sane a person named G. M.?

A. Yes.

EXTRACTS FROM EXPERT  
TESTIMONY, AND CROSS-  
EXAMINATION THEREON,  
AS GIVEN FOR DEFENDANT.

DEPOSITION OF DR. E.

Examined by Mr. S. L. D. H., of  
counsel for defendant.

Q. Are you a practising physi-  
cian and surgeon?

A. Yes.

Q. What is your position?

A. I am superintendent of the  
——— Hospital for the Insane at  
———.

Q. That is the public hospital for  
the insane?

A. Yes.

Q. Did you examine G. M., who  
has been referred to in this case?

A. Yes.

Q. What was the date of your  
examination?

A. December 17.

Q. December 17, 1913?

A. Yes. Together with Dr. B.

Q. In what condition did you find  
him? Was he suffering from any  
trouble?

A. Yes. I found him suffering  
from melancholia.

Q. In view of all the evidence  
which has been given at this trial,

Q. How long has he been incarcerated there?

A. I was absent in Europe at the time he came in, but according to our records he came in on the 15th of July, 1912.

Q. You were absent at the time?

A. Yes. I first saw him, I think, on the 27th of August.

Q. Did you examine him personally on your return?

A. I did. Either the first or second day after my return.

Q. Did you have a report with regard to him when you returned?

A. I had the case book reports.

Q. What was the condition of G. M. when you personally examined him?

A. I found him suffering from melancholia.

Q. Is that a technical expression?

A. It is the ordinary technical phrase for that form of mental disorder. Of course there are any number of subdivisions of that form. If I had to put it down before a lot of medical men, I would say he was suffering from involutinal melancholia.

By the Court:

Q. Does that mean melancholy against his will?

A. No, sir. Evolutional is a gradual progress forward. Involutinal is a progress backwards.

By Mr. S., continuing:

Q. Is that a form of mental disease?

A. Decidedly.

Q. Would you call it insanity?

A. Certainly.

Q. Will you describe to the court the characteristics and peculiarities of this form of mental disease?

A. Melancholia, especially this form, is a disease of very slow

and the documents which have been filed in this case, what is your conclusion as to the sanity or insanity of G. M. during the months of December, 1911, January, February, March, 1912?

A. In December, 1911, and January, February, March, 1912, I do not consider he was insane at that time.

Q. Do you consider he was sane?

A. Yes.

Q. On this evidence and on the documents filed, what in your opinion was the mental capacity of G. M. to appreciate the nature of the transactions referred to in this case? That is to say, on or about the 5th of March, 1912, and about the 21st of February, 1912?

A. I think he had the mental capacity to fully appreciate the nature and quality of his act at that time.

Q. You say you think—is that your opinion?

A. I give that as my professional opinion.

Q. Would you consider from the evidence made in this case, and from the documents filed, that Mr. G. M. was suffering from confirmed melancholia (which is a continuous and progressive disease) during the months of December, 1911, January, February and March, 1912?

A. No.

Q. With regard to the evidence of Dr. S. and Dr. T., what have you to say?

A. I have gone very carefully over the evidence of Dr. S. and Dr. T. and studied their descriptions as carefully as I possibly could. Dr. S. had first observed Mr. M.'s condition in the beginning of the year. He said he was agitated and that

growth, ordinarily speaking (I am speaking, of course, of cases that come into hospitals). The disease exists for months and months prior to the admission of the patient. The beginning of the disease is so insidious that very often the friends do not notice it in the early stages at all. After that it is gradually progressive. The disease in many cases (especially in involutional melancholia) is dependent upon a condition that we call arteriosclerosis—that is, hardening of the arteries—and that disease is progressive beyond a doubt. At the beginning the symptoms are very insidious, and perhaps the friends do not notice it at all. They gradually become worse and worse, and finally the man has to be taken in charge. After he is taken in charge he still continues to grow worse. Finally he winds up in a condition of what we would call dementia, which is followed by death.

Q. Is there any going back, or recovery?

A. I think not. Not in a case of arteriosclerosis. You cannot soften the arteries when they are once hardened up.

By the Court:

Q. Then, it is a result of a physical condition?

A. A physical condition, probably plus some worry as an exciting cause. The thing depends in the start upon a physical condition—a condition of the arteries.

By Mr. S., continuing:

Q. And that is what G. M. had and has?

A. Yes; he had and has.

Q. Would you say that was the cause of his mental condition?

he had insomnia. These are the two principal points he brings out. He made no mental examination, and so he declares. He prescribed nothing for him except some bromide. Then, coming on toward the end of March (he had seen him during these months and had made no examination), I realize fully the statement he makes that he advised Mrs. M. to send him to a sanitarium and he stated that he took this action on the advice of Mrs. M. He is quite careful to state that in his evidence.

By Mr. S., of counsel for plaintiff:

Q. Now coming to Dr. T.'s evidence?

A. Dr. T. sees him in March, presumably the week of the 5th of March, or, say, during the first two weeks of March. He also states that he made no mental examination. His examination was directed to Mr. M.'s physical condition rather than to his mental condition. He states that Mr. M. had an idea that he had lost his property and that he had ruined his son. I cannot quote the exact words, but this would be the substance of it. Dr. T. states that he cannot tell whether this condition was permanent or transitory, but he makes the very significant statement later on that the case developed during the months of May and June. Now these are the statements of Dr. S. and Dr. T., and these statements go to show that the man was depressed, suffering from worry, which is quite normal under the circumstances. I attribute this, of course, to the fact that his son was ill, and that the property of the son—quite large amounts as given by the evidence—was in a somewhat

A. I think so. As I say, that probably plus some worry. I don't know what it would be at all. The condition of arteriosclerosis tends toward mental enfeeblement.

Q. Inevitably?

A. Inevitably.

Q. Insanity, I presume, has many different forms?

A. Oh, many.

Q. Are they classified at all?

A. They are. Too much so, very often.

Q. When you use the expression, "melancholia," that is one of the classifications?

A. Yes, it is one.

Q. It is the one into which you put this particular case?

A. Yes. As I say, there are several forms. You might divide melancholia into a number of forms, but, ordinarily speaking, it is melancholia.

Q. It is that form of insanity which is known among scientific men as melancholia?

A. Yes.

Q. How does it affect a patient with regard to his capacity for doing business?

A. I should think it would affect him deleteriously.

By the Court:

Q. What do you mean by "deleteriously?"

A. That is giving a general opinion. I think any man suffering from melancholia could not make as fair a judgment as an average man. Of course, I cannot say anything at all positively as to Mr. M.'s judgment prior to my seeing him. I can only judge that this disease had made slow progress, and, in my opinion, must have existed for

embarrassed condition. It is only normal that a man under these circumstances should be in this condition of worry.

Q. What conclusion do you draw from the evidence of Dr. S. and of Dr. T., presuming that all the facts they state are correct?

A. The conclusion I draw from the evidence of Dr. S. and Dr. T., after giving every possible consideration to it, is that during the months of January, February and March, 1912, Mr. M. was suffering from worry and anxiety, due to the condition of his son and his son's affairs, and that he was not insane.

Q. Assuming that Mr. G. M. did make the statement on different occasions that his family was ruined, and his son was ruined, and that he was ruined, prior to the first week of March, 1912, does that, in your opinion, indicate that he was insane and incapable of appreciating any of these transactions with W. M.?

A. No, I don't think so, because it is quite natural that at these times he would be suffering from extreme depression and anxiety with these facts in his mind. He might very well feel, and very naturally feel, very much depressed and would therefore probably give expression to rather exaggerated ideas of depression.

Q. Presuming that on one occasion, about the end of February, Mr. G. M. did make a statement to his wife that the best thing would be for the three of them to die together, does that, in your opinion, indicate that he was suffering from continuous and progressive melancholia, and that he was unable to appreciate his actions?

months, or probably a year or more, before he came into my care. Of course, that is only a supposition. I can only speak of the man as I found him.

Q. Do you conclude from the condition in which you found him that that disease must have existed for a year?

A. For months before.

Q. Six months?

A. I should think more. I do not think that that condition of arteriosclerosis could have originated in six months.

Q. During that time—six months—would that patient be in an ordinary, normal condition to transact business?

A. Ordinarily speaking, I should think not. In this special case, I cannot say.

Q. What has been the conduct of the patient since your return—can you speak of that personally?

A. He has been a case of melancholia, with all manner of delusions, to the effect that he was ruined, and his family was ruined; that he was to be arrested and put in prison, and that he was to be tortured in all sorts of ways. These were the original symptoms when I saw him first. Prior to that, of course, there was a number of things of which other witnesses can speak. I am only speaking of my own personal knowledge. That was his condition when I first saw him, and that condition has continued, and continues to-day.

Q. Does it get better or worse?

A. If anything, worse. There is a tendency towards what we call "feeble-mindedness."

Q. According to your experience and observation, at the time you

A. No. I would not say that. He might make that same statement if he was quite depressed, owing to the condition of his son. If he felt that his son was dying, he might make use of the expression that it was better for all three of them to die together. However, you must follow that by his actions subsequently. He had plenty of opportunity during the following month, if he was determined to suicide, to do so. That would be before he was interdicted, but he did not do so, as far as I can read the evidence.

Q. What I want to know is, if a person suffering from the disease which you found G. M. suffering from, can at times or on certain subjects behave rationally and talk rationally?

A. On certain subjects? Do you mean on certain subjects or rationally at all, at any time?

Q. I mean can he conduct himself rationally, eat, drink, and go to bed, and do all that kind of thing, like an ordinary person?

A. If a man has a condition of melancholia, while he is ill with that disease, he does not conduct himself rationally on all subjects, no.

Q. I did not say on all subjects. I said on any subject?

A. Yes. My answer is that a man with melancholia may intelligently discuss many things.

Q. Is it not a fact that patients suffering from melancholia and depression and delusions, as you have described it, might deceive an ordinary person as to their condition, or might dissimulate their condition, and deceive persons who are not experts as to their delusions?

A. You are quite contrary to the facts. They magnify enor-



first saw the patient, had the disease made marked progress?

A. My conclusion, when I first saw the man, was that the disease must have existed for quite a lengthy period beforehand. I could not just say how long. Of course, I might be wrong, but that would be the conclusion I would draw from the condition of the man—that the disease must have existed over months and months previously. That is the conclusion I would draw as a medical man.

Q. According to your observation, for what length of time had the mental condition of the patient existed at the time you saw him?

A. It is impossible to say definitely, but in my own opinion (although I cannot say definitely) he must have been insane for months before. In my opinion, I have no doubt of it. Of course, I cannot say positively. It is only an opinion.

By the Court:

Q. That is, months before you saw him in the month of August?

A. Months before I saw him in August. Of course, I could not say any definite, fixed time.

By Mr. S.:

Q. Is that according to your experience?

A. That is according to my experience.

Q. I presume you have had experience in many similar cases?

A. Probably some thousands.

Q. Is it according to your experience that the disease with which G. M. is afflicted is continuous and without intermission?

A. It is continuous and progressive—both according to my own observations, and from the weight of

mously their condition, and their hypochondriasis. They never hide their condition. They do not try to hide their case at all. They give it away too much.

By the Court:

Q. At the present time, can this man converse with apparent good sense on some subjects?

A. On some subjects. For instance, he talked to me over the early history of his life in N. B., and told me how he tramped the rivers from there, and he knew a great deal about the geography of it. He told me, to my surprise, that his son, R. M., was a cadet at the K. Military College when he learned that I was from K.

all authorities I have read at different times. It is continuous and progressive.

Q. How does arteriosclerosis affect the brain?

A. By the changes in the circulation. The smaller blood vessels become thickened and the larger ones become what we call calciform—that is, deposits such as lime come in the coats, and they get thickened, so that the circulation in the brain is markedly affected, and this increases all the time; finally, if the patient lives long enough, it leads to what we call dementia.

In rebuttal:

Q. Dr. U., you have already been examined in this case?

A. Yes.

Q. Were you present yesterday when the medical experts for the defense gave their evidence?

A. Part of the time. I may say I have not read any of the evidence at all, thank the Lord. I did not have to. I heard part of the evidence yesterday.

Q. The opinion was expressed yesterday by one of the medical experts for the defense that it was not possible to discover by examination of the patient whether he is suffering from cerebral arteriosclerosis or not. Do you agree with that? You understand, I do not mean any patient in particular?

A. I do not agree with that entirely. For instance, if I find a man with marked arteriosclerosis in the radial arteries, there is a strong suspicion in my mind that that arteriosclerosis extends more or less all over the body, including the brain. If I find, then, there is marked arteriosclerosis of the tem-

poral arteries, then I am still more confirmed in my belief. Then, if the man has attacks of dizziness—almost semi-paralytic attacks—I would not hesitate to diagnose cerebral arteriosclerosis. I might be wrong, but that would be my opinion as a medical man.

Q. Suppose cerebral arteriosclerosis does exist, does it constitute an organic lesion of the brain?

A. Certainly. Not of the brain substance, but of the brain as a whole.

Q. What is the effect of that?

A. The effect is to weaken a man's intellect all through.

Q. Would that be a curable condition or an incurable condition?

A. Absolutely incurable.

Q. There is no going back?

A. No going back.

Q. It has been sworn to here, in the medical evidence for the defense, that the condition of a patient suffering from melancholia is at all times apparent, and that it is impossible for the patient to conceal it or dissimulate it. Do you agree with that or not?

A. I do not. I could cite you a score of cases of marked melancholia confined in an asylum where I would defy anyone, for days, to say it was a case of melancholia. I do not agree with that opinion at all.

Q. Are you speaking now from your personal experience in the asylum?

A. That is my personal experience, extending over forty years, and covering perhaps 8000 or 9000 cases.

Cross-examined by Mr. G.:

Q. Do you suggest that this particular melancholiac G. M. dissimulates?

A. No; I don't think so.

Q. And that was the meaning of your answer to Mr. S.?

A. That is the meaning of my answer to Mr. S. He might appear to be sane for a time, but no insane man can imitate sanity. If I may be allowed to say it, an insane man might pass muster for a time as a sane man. I can cite you dozens of cases.

By the Court:

Q. Could a man suffering from melancholia meet old friends, for example, and be able to converse with them about the affairs of his childhood, and of his former life, in apparently a perfectly rational way?

A. It is quite possible. I have known a number of cases of the kind in my own experience. Of course, he could not do it for any extreme length of time, but I have known cases of melancholia go for a day or two, or perhaps a week, brightened up so that they would be apparently sane. However, at the same time they were insane.

Q. With a melancholiac there is usually some delusion or apprehension?

A. Usually.

Q. When there is such a delusion, is it the tendency of the melancholiac to give vent to that delusion in speaking of his friends and companions?

A. I think the tendency is that way. On the other hand, the melancholiac sometimes has the power to restrain himself to a large extent. Probably his will power is not lost entirely. Of course, the tendency is to talk freely about his delusions, but, if something else comes to his mind, he has the

power to control himself to a certain extent and not give expression to the delusions. I have seen scores of cases of that kind. As I say, I have probably had 8000 or 9000 or 10,000 cases of insanity to deal with in my experience, and I know they can control themselves to a certain extent, and appear absolutely sane.

DEPOSITIONS OF DRS. V. AND X.  
DR. V.

Examined by Mr. H. J. E., of counsel for plaintiff:

Q. How many years have you been practising medicine, Dr. V.?

A. Since 1883, 30 years.

Q. I understand you have made a specialty of mental diseases?

A. I have.

Q. How long have you been engaged in that special work?

A. About twenty-six years.

Q. You were connected with one of the hospitals for the insane here in Montreal?

A. I am.

Q. Which one?

A. The ——— Hospital.

Q. Is that a large hospital?

A. It is.

Q. About how many patients would you have there under treatment at a time?

A. I was looking up the records the other day, and we had altogether, public and private, about 2300 cases.

Q. Did you have occasion to visit and examine G. M., now confined in the Verdun Asylum?

A. I did.

Q. When did you examine him?

A. A week ago last Sunday. November 30, I think it was.

DEPOSITION OF DR. D.

Examined by Mr. S. L. D. H., of counsel for defendant:

Q. What is your present position?

A. Superintendent of the ——— Sanitarium, ———, Ontario.

Q. What has been your experience with mental diseases?

A. I graduated in Toronto in 1890. I spent two years in general medicine, and from that time on I have been continuously in mental work. I spent ten or eleven years in ——— Asylum, and the balance of the time at ———.

Q. What sort of people do you treat at ———?

A. At the ——— Asylum we treated all mental diseases. At the ——— Sanitarium we have all classes of patients.

Q. What is the size of the ——— Asylum?

A. The ——— Asylum has about 1000 or 1100. In the ——— Sanitarium we have a capacity of about 130.

Q. I understand you examined G. M. a short time ago?

A. December 11, 1913.

Q. Did you find him suffering from any mental disease then?

A. I found him suffering from melancholia, with delusions.



Q. Will you please describe to the court the condition in which you found him on your examination?

A. I found him suffering from profound involuntional melancholia. His depression was very, very marked. He was overcome by an intense idea of ruination. I found his memory very much affected. He could not tell me the date nor the year. He could not tell me how long he had been in the institution. He was under the impression that his wife was in an adjoining part of the institution and he had certain expressions that he made use of in the course of the conversation quite characteristic of melancholia of involuntional, such as "My God"! "My God"! At times in all of these cases, even in the advanced stages, there is a certain amount of coherency of thought and they can answer questions and their answers taken by themselves are quite coherent.

Q. From your examination at that time, and from your examination alone, were you able to form any opinion as to the date when this disease had its beginning?

A. Taking his case, independent of any legal connection, but in the ordinary run of my experience, I should say from the enfeeblement I noticed in his memory I would naturally come to the conclusion, as I have in many other cases which I have observed, that the disease had an antecedent history of quite a duration of time.

Q. Have you been present in court during the hearing of this trial?

A. Yes.

Q. Did you hear the evidence of Dr. W.?

Q. You have, I understand, read all the evidence which has been given at the trial?

A. Yes. I heard part of it and I read it all.

Q. From the evidence and from the documents filed which you may have read are you able to form an opinion as to the sanity of Mr. G. M., the father of the plaintiff, during the months of December, 1911, and January, February, March, 1912?

A. I consider he was sane.

Q. During these months?

A. During these months.

Q. Basing yourself on the evidence and the documents filed, what in your opinion was the mental capacity of G. M. to appreciate the nature of his transactions with Mr. M. which are referred to in this case?

A. He was mentally capable of doing it.

Q. That is your opinion?

A. That is my opinion.

Q. Do you mean that he was naturally capable of appreciating it?

A. Yes.

Q. Would you consider from the evidence you read and from the documents you read that G. M. was suffering from confirmed melancholia (which I understand is continuous and progressive) during the months in question?

A. No.

Q. What are your reasons briefly for the opinions you have expressed regarding G. M.'s condition during these months of January, February and March?

A. Well, as from the evidence as given by Mr. W., his solicitor, who

A. I did.

Q. And Dr. S.?

A. Yes.

Q. And Dr. T.?

A. Yes.

Q. Assuming that the facts recited by these several witnesses are correct, have you in these facts a basis upon which to form an opinion, and if so, will you state to the court what that opinion is as to the date of the commencement of this disease in Mr. M.?

A. Assuming these facts to be true, I am of the opinion that the man was insane for quite a period of time prior to his commitment to the Verdun Asylum. I would be of the opinion that the man was insane in the month of January or February positively.

Q. 1912?

A. 1912.

Q. What is the character of this particular disease? By what name would you term it?

A. In later years we have adopted for the time being only, as a quasi classification of insanity, the German classification, and such conditions as are observed in the case of Mr. M. we place in the category of melancholia of involution. This is a disease characterized by uniform depression, accompanied with fear. It is, however, accompanied by distinct delusions, self-accusations, and self-depreciation of a persecutory nature and of a hypochondriac nature, with disturbances in the train of thought, and in the vast majority of cases it ends up with deterioration of the brain. There is a certain percentage of cases which are considered to recover, but they are very small.

saw him frequently in these months—between December and March—and who transacted business for him. Mr. W. drew up certain documents, and he states in his evidence that Mr. M. insisted on and directed certain changes in these documents, and that he was capable of understanding them. Mr. W. also says that he did not notice anything unusual in the man at the time. That is one of my reasons. Another reason is that he performed certain business transactions during these months, as evidenced by the deposition of Mr. D., who told us what Mr. M. had done with reference to the dredges. This is also evidenced by Mr. B., whom Mr. M. advised in regard to some farm lands, and by the evidence of Mr. P. in regard to the purchase of some real estate in Montreal. I consider all this evidence is good evidence of the man's mental condition and capability of doing business at that time. He also met many witnesses upon the streets in Montreal, who knew him well. He met them in the different hotels, the Corona, the St. Lawrence Hall, the Windsor Hotel, and so on. They had conversations with him and they stated that they noticed nothing out of the way in his actions or speech. A little more important is the evidence of men like Mr. S. and Mr. J., both capable business men, who impressed me here in the witness box as being straightforward business men. They saw Mr. M. frequently during the same months and had conversations extending from ten minutes to an hour with him at different times. They knew him intimately and they stated that they

Q. From the history of this case, do you find any evidence of intermission in the disease, or was it continuous and progressive?

A. In my humble opinion there was no intermission; simply a progression of symptoms.

Q. So that, assuming as a matter of fact that this disease existed in January (or certainly in February), in your opinion was the man insane from that time on?

A. It is my opinion that from that time on he was insane.

His Lordship: From what time on, Mr. E.?

Mr. E.: From the month of January, 1912, my Lord.

By the Court:

Q. Is that what you mean, Dr. V.? Is it your opinion that he was insane from the month of January, 1912?

A. Yes, my Lord; from towards the close of the month of January.

Cross-examined by Mr. H., of counsel for defendant:

Q. Will you tell us exactly what you mean by "insane?"

A. I would say that the man was deprived of the power of exercising his common sense.

Q. You consider he was deprived of the power of exercising his common sense since January?

A. I would say that he was deprived of the exercise of his common sense. I might specify more clearly by saying in matters involving the higher relations in his life.

Q. What do you mean by the "higher relations?"

A. Passing judgment upon matters of importance to himself and those connected with him.

Q. Will you tell us exactly what were the facts upon which you base

saw nothing wrong with him in his actions or his reasoning power. They did not notice anything out of the way. Then, evidence is given of certain documents or letters that he wrote in reference to business matters during these months. These documents show fairly clear reasoning. We also have the evidence of Mr. S., an independent lawyer, who occupied adjoining offices to Mr. W. Mr. S. saw him frequently during the same months. He did not have any business transactions with him whatever. I think he was absolutely independent in his opinion. He stated he saw nothing wrong with Mr. M. during that time. In the converse we have the evidence of Dr. S. and Dr. T. and the sanitarium nurses, who saw him during the same period at certain intervals. They stated he was very much agitated, suffered from insomnia and they noticed a number of conditions which I consider were, at that time, the natural anxieties of a man suffering from stress and worry. That is my own evidence in regard to his condition. He was anxious and worried over his son's business and his own affairs at the time.

Q. Assuming that on certain occasions prior to the first week of March, 1912, or about that time G. M. made statements to the effect that he was ruined or that his family was ruined or his son was ruined (as stated in the evidence), would that in your opinion indicate that he was insane and incapable of appreciating the nature of these transactions with Mr. M. and Mrs. M., referred to in this action?

A. This man was under great stress and strain at the time, and,

the opinion that he was insane in January?

A. In the first place, I base myself upon the fact that in melancholia there is a delusional state which differs from the delusional states observed in other forms of insanity. That is to say, the delusional state of ruin or depression is of such a character and such an intensity that once it takes a hold of the individual attacked it dominates his whole psychic being, even without there being anything in his external appearance or in his conversation to cite the fact in particular. While I do not feel that any close observation, such as we ordinarily make of a patient coming under observation, was made, still there was a sufficient examination made, and the description of the examination as given by Dr. T. was a very classical presentation of a case of melancholia of that character, as it would appear in the wards of our asylum. The manner in which Dr. T. described his interview with the patient, his downcast appearance, his state of torpor; when put a plain, ordinary, sensible question as to what was the matter with him he could not describe it except as that of a condition of ruin. The very words he used are typical of these cases—"It is terrible." "It is terrible." These are the words that a melancholiac would use. It is a vague expression, if you will, of the profound disturbance of his mind.

DEPOSITION OF DR. X.

Examined by Mr. L., of counsel for plaintiff:

Q. For how many years have you been practising your profession?

I believe, was in a fair way to lose a large amount of money. It appeared to the man, who had worked very hard in times gone by, that this looked as if the commencement of ruin was upon him, and that it might go on. He expressed these delusions especially when he was in the hospital, in contiguity to his son, who was very ill. I consider these were normal anxieties at that time.

Q. In giving your opinion, do you bear in mind the statement he made on certain occasions as to ruin, or the statement made by Mrs. M. that on one occasion he said the best thing would be for the three of them to die together?

A. I think in his normal mind he might give expression to a thought of that kind when they were suffering. At a certain time he might give expression to the idea that the best thing to do would be to die. I have known normal men give expression to the thought that they wished to God they were out of the world, or wished they were dead, or something like that.

Q. You have borne these facts in mind in giving your opinion?

A. I include them together with the fact that he met these intelligent people at different times on the street and never expressed these ideas to them at all, which, undoubtedly, he would have done if the disease of melancholia were established at that time.

Cross-examined by Mr. S., of counsel for plaintiff:

Q. May a person be insane with that form of insanity known as melancholia and yet perform the usual acts of life in the usual way?

A. It depends again upon the delusion that he may have.



A. Since 1889.

Q. Have you made a specialty of the study of certain diseases?

A. Yes, sir. Since 1894 I have been occupying the position of Medical Superintendent of the ——— Hospital for the Insane.

Q. How does that hospital compare with others in the Province of Quebec for importance?

A. I think it is the largest asylum in the Province of Quebec, and I think about the second largest in Canada, if not the largest.

Q. You have been there since 1894?

A. Yes.

Q. Did you ever examine Mr. G. M., who is in question in this case?

A. Yes, sir.

Q. Will you say on how many occasions?

A. I examined Mr. M. on the 28th of February last, 1913.

Q. At the request of whom?

A. I examined him at the request of Dr. Y., who sought my opinion as to Mr. M.'s condition at the time, and as to whether I could form an opinion in regard to his condition for some time previous to that.

Q. Let us deal with the marks whereby you recognized this form of insanity which you call melancholia. What were these marks?

A. At the time I found Mr. G. M. a patient in the Verdun Hospital for the Insane. He was in a great state of anxiety and was suffering from delusions of a depressing and terrifying nature. He was also suffering from hallucinations of hearing, also of a depressing and terrifying nature.

Q. Starting from what you found on the 28th of February, and adding

Q. If we leave aside the question of the remissions and come back to the question I asked you, I would like you to inform the court whether persons recognized to be insane with that form of insanity known as melancholia may perform all the usual acts of life in the usual way?

A. Again I must answer it depends upon the intensity.

Q. I am not speaking of the intensity at all. I am speaking of persons who are recognized as being insane?

A. I am speaking of the same people.

Q. So that, according to you, persons recognized to be insane from melancholia may perform the usual acts of life in the usual way?

A. Under certain circumstances.

Q. I am speaking of any patient suffering from melancholia. I am not speaking of all patients.

A. It depends on the intensity. In the remissions the disease still exists, but at these times they may, and do, direct one in carrying on certain business, in ways which are perfectly justified and perfectly clear.

Q. During these remissions, are they sane or insane?

A. They are insane; the depression still exists.

Q. Is it not a fact, Dr. D., that a patient so afflicted may converse for a considerable length of time without betraying his condition?

A. Not in my experience. They worry the life and soul out of me every day. It is hard to get away from them. They will persist in talking about their delusions.

Q. Is it not a fact that in an unlimited number of cases the patient



to your own findings those of Dr. S. and Dr. T., at the respective dates they have mentioned, and also the evidence of Dr. U. and Dr. W., would these findings and these facts constitute a previous history of the case sufficient to complete your conclusions as to the duration of time during which this disease had pre-existed?

A. Yes, sir.

Q. Taking these findings and these facts in conjunction with your own observations on the 28th of February, what conclusion have you reached as to the time during which this disease probably pre-existed?

A. I would date it at least as far back as December, 1911, and would say that it was continuous and progressive since that time.

Q. When you say "progressive" I suppose in matters such as this it is merely a figure of speech, because it is progressive the wrong way. It is an aggravation really?

A. Yes. "Progressive" means aggravation.

Q. Now, Dr. X., you are not a business man?

A. No. I have no time for that.

Q. But you have made contracts. Apart from the contract of marriage, of course?

A. I never made a contract in my life. I never did anything but study medicine.

Q. You know what a contract is?

A. Yes; sure.

Q. Would you believe that a man laboring under this disease of melancholia, the presence of which was detected as far back as December, 1911, and which became graver up to the month of March, 1912, would

will converse for a certain length of time without referring to his delusions at all, unless his attention is called to them? That is to say, as long as you keep the patient on indifferent subjects he may be perfectly sane, to all outward appearances, but as soon as you approach him on the subject of his delusions then he is persistent and the delusions become predominant and paramount?

A. My experience is, with friends constantly visiting the patients, they are always harping on their delusions. The friends are constantly telling me, "Well, So and So will persistently talk about his ruin," or whatever the delusion may be. My experience has not been with strangers, but with the friends of the patients. These people persist in talking to their friends about their delusions.

Q. So that you would give it as your definite opinion that patients afflicted with melancholia must always betray their condition to persons with whom they are in conversation?

A. Particularly friends and relatives.

Q. According to your experience, how long would the disease have existed in this man before he was committed on July 15?

A. In May and June. I agree with Dr. S. and the evidence of Dr. T., and the fact that in that month he noticed a change.

Q. So that, in your opinion, he was insane as far back as May, 1912?

A. Yes; the end of May.

Q. You will not go back any further than that?

be able to exercise his judgment and mental faculties for the purpose of transacting business, or entering into contracts?

A. No; certainly not.

Cross-examined by Mr. G., of counsel for defendant:

Q. Will you please specify to me the facts which you have seen in the evidence which have induced you to say that in December, January and February it was a case of confirmed melancholia?

A. I find there most of the symptoms which I found myself in February, 1913, and which gave me the opinion then that it was a confirmed stage of melancholia.

Q. I am asking you to specify these symptoms, which, according to what you understood or read in the evidence, existed in December, 1911, and January and February, 1912?

A. We find the man to have been in a great state of moral pain, anxiety and anguish, and that he had delusions that he had ruined himself and family. He was acting in a very excited manner and speaking incoherently, according to the evidence. This condition is stated to have existed in December, 1911. Then, we find these ideas repeated at frequent intervals by those who observed him in January, February, March, April, June, July, and so on.

#### DEPOSITION OF DR. Z.

Examined by Mr. S., of counsel for plaintiff:

Q. You are a practising physician in the City of Montreal?

A. Yes.

A. I cannot find evidence that would commit the man further back than that.

Q. Is the disease of melancholia one of rapid development or of slow development?

A. It depends on the case. Sudden shock will bring a patient into melancholia in a short time.

Q. He cannot be sane and insane at the same time of course?

A. No; he cannot be water and oil.

#### DEPOSITION OF DR. A.

Examined by Mr. S. L. D. H., of counsel for defendant:

Q. You are practising your profession in Montreal?

A. Yes.

Q. Since how long?

A. I graduated in 1901 in medicine and in 1897 in arts at McGill.

Q. Have you been practising in Montreal ever since?

A. No. I was two years at the — as house surgeon; then I went to the — Hospital in Baltimore for the better part of a year. Then I went to Europe and I had a year in Switzerland with Professor Von Munerkof, working in his laboratory. Then I was in various parts of Germany, Berlin, and so on. After that I went to Paris. Then I spent a year in London, at the National Hospital for the Paralyzed and Epileptics, where they make a specialty of diseases of the nervous system.

Q. If I understand you rightly, you have made a specialty of nervous diseases?

A. Nervous and mental diseases; yes.

Q. You visited Mr. M. at the asylum?

A. Yes.

Q. In what condition did you find him?

A. I have a letter which I wrote to Dr. Y. I think if I were permitted to read that letter to the court it might save time and convey all to you that I could convey by my evidence.

Q. Was the letter you are about to read written after your visit?

A. Yes. I wrote it that same evening.

The letter in question reads as follows:

MONTREAL, May 3, 1913.

MY DEAR DR. Y.: Re Mr. M., whom I examined in your presence at the Protestant Hospital for the

Q. Have you specialized in any particular branch of medicine?

A. Nervous and mental diseases.

Q. What is your experience in that regard?

A. I am lecturer on nervous diseases at — University. I am neurologist at the — General Hospital. I am consulting neurologist at the Hospital for Insane, —. I am professor of nervous and mental diseases, University of —, and have been for over twelve years. I was associated for some time with the — Hospital for the Insane. I am consultant for the Hospital of Mental Disease, —, Vermont. I was associated for some time with the Hospital for the Insane at —, England. I have been employed by the United States Government for many years as an expert for the criminal insane in the State of — and the State of —. I am neurologist for the — Railroad. I am a member of the — Neurological Association. I am a member of the — Association. I am a member of the — Institute for Criminal Law and Criminology.

Q. How long have you been practising your profession?

A. I graduated twenty-eight years ago.

Q. From where did you graduate?

A. — University, —.

Q. You say you are consulting physician at — Asylum. Is that the — for Insane at —?

A. Yes.

Q. With which Dr. U. and Dr. W., examined here on behalf of the plaintiff, are connected?

A. Yes.

Insane at Verdun, the 2d inst., I find that this patient is suffering from great mental depression, associated with delusions of a persecutory nature. Suicidal tendencies are evidently marked, also he has made more than one attempt on his life. He imagines that he has ruined his whole family by giving away money and also some contract that his son had. He has, he thinks, committed the great sin—he is lost. When asked why he gave away his son's money he answered that he did not know; he must simply have got a notion. He could not remember just when or where he had done it. Instead of a shrewd business man, his mind is now full of indecisions. He practically never makes a positive statement, and seldom ever gives a positive answer to a question. He has insane delusions and imagines that people are saying things against him of a horrible nature. He stated that everything he said to us was overheard because the room was wired and connected with telephones and all our remarks were noted down already somewhere. Examination showed paralysis of the movements, atrophy of the optic nerve, and blindness of the right eye, the result of an attempt at suicide with a pair of scissors. He has all the evidence of marked arteriosclerosis. I am, therefore, of the opinion that this patient is suffering from mental disease of the nature of an involuntional melancholia, associated with cerebral arteriosclerosis. In answer to the question as to whether this would have had bearing of an etiological nature on his peculiar action in giving away the money, one can only answer that it probably

Q. You are consultant for mental diseases at that hospital?

A. Yes.

Q. In your practise at the hospital and the different places you have described you confine yourself to nervous and mental diseases?

A. Yes.

By the Court:

Q. Has your work as a specialist been running for many years?

A. About eighteen years.

By Mr. H., continuing:

Q. I understand you have had occasion to examine G. M., the father of the plaintiff in this case?

A. Yes.

Q. At the Verdun Asylum?

A. Yes.

Q. On what date did you examine him, and what did you find him suffering from?

A. I examined him on the 11th of December, 1913. He was suffering from melancholia.

Q. You have read through the evidence given on both sides of this case?

A. I have.

Q. You have also read the letters filed as exhibits from G. M. to W. M., the defendant, and from Mrs. M. to M., as well as the other writings of G. M. filed in this case?

A. I have.

Q. Are you able to form any opinion, as a result of the evidence given and the documents filed as to the sanity or insanity of Mr. G. M. during the months of December, 1911, and January, February and March, 1912?

A. I believe he was sane.

Q. Is that your opinion as a medical man?

A. That is my opinion as a medical man.

did, but one would need corroborative evidence of some change in character, some peculiarity of ideas and actions present at the time. One of course recognized that the symptoms of cerebral arteriosclerosis may often come on almost acutely, following some shock either mental or physical, or as a result of mental strain. I would, therefore, urge the necessity of searching for corroborative evidence of the nature referred to.

With kind regards, I remain,

Yours sincerely.

Q. Did you or were you able to form an opinion as to how long the condition in which you found the patient had lasted?

Witness: Do you mean at the time of that examination or since?

Counsel: At the time you made the examination?

A. I could form no opinion from what I saw, except that it must have been present for some time. One could not say more than that.

By the Court:

Q. What would "some time" indicate? "Some time" is a general expression of course. As regards weeks, months, or years, what would you want the court to understand?

A. One could definitely say it had existed for some months I think; especially if one could take into account the history one got with the patient one could very positively say it had existed for some months.

Q. From what you knew at the time you went there to examine him?

A. From my examination of Mr. M. at that time I would say that the condition might have lasted for a

Q. And as a specialist?

A. And as a specialist.

Q. Basing yourself on the same evidence and the same documents, what is your opinion as to the mental capacity of G. M. to appreciate the nature of the dispositions of his property in question in this case, about February 21, 1912?

A. I think he was a sane man and perfectly capable of doing it.

Q. You consider he was capable of appreciating the nature of these transactions?

A. I do.

Q. Would you consider from the evidence and documents on record that Mr. G. M. was suffering from confirmed melancholia (which is a continuous and progressive disease) during and throughout these months?

A. He was not.

Q. Will you give us briefly some of the reasons for the opinions you have expressed in regard to Mr. G. M.'s mental condition?

A. To make it brief, I may say that I agree thoroughly with Dr. E. and Dr. C., Dr. D. and Dr. B. I agree with what they have said in connection with the different witnesses who came up from Dalhousie and who were residing in the neighborhood of Montreal.

Q. Do you agree with the reasons they expressed with regard to their opinion as to the mental condition of G. M. during the time in question?

A. Yes. I also agree with them in their opinion as regards the nurses in the sanitarium—that at no time was a delusion demonstrated or ever shown to be a fixed delusion. I think that, as the court



couple of years of it might have lasted for a less period. One could not make a statement, as I said in that letter, as to how long this had lasted. One could not judge from one's examination.

Q. Did you hear the evidence which had been given in this case by other specialists and by those intimate with the patient?

A. I heard Dr. W.'s evidence and I heard the evidence of Dr. U. I also heard the evidence of Dr. S. and Dr. T.

Q. Does that enable you to form any more definite opinion with regard to the time this disease may have lasted?

A. I think it does; yes. Dr. S. gave evidence that in January the man was suffering from insomnia, mental indecision, and that he was worrying and showed lack of concentration. This was in January. Now, in February and March Dr. S. recommended that they should send him to a sanitarium for mental diseases. Evidently he had progressed backwards a good deal. In June Dr. S. had to bring him home from the corner of Peel street. This man was then standing in the middle of the street in a state of mental confusion. On July 11 he had definite delusions. Then, as far as I know, he was a man quite well off, but at the same time he was going around asking everybody for ridiculous positions. I mean to say he was asking for positions that were ridiculous, taking into account his position and his age. Imagine a man of his age volunteering or thinking of climbing telegraph poles. He was a man who had managed big business affairs,

stated yesterday, we may apparently be taking sides, and to offset this, I believe we should lay particular stress on that part of the evidence where no outside influence can be brought into play. That in particular would be the letters written by Mr. M. during that period. They speak for themselves. These letters relate to certain business transactions which have been shown to be carried out and which were, in my opinion, carried out in a very correct manner. If G. M. was able to undertake journeys, considerable distances from Montreal and carry out business on these journeys—if he was able to go, in the spring of the year, some hundreds of miles alone, down to Dalhousie and come back again, to my mind this clearly demonstrates that the man used his will power and his judgment and nothing in his conduct in any way showed that he was suffering from an insane condition. Dr. S., who saw him in December and examined him for the first time in January, says that at that time he was suffering from nervousness, agitation and depression. At that time Dr. S. did not see any symptoms of insanity. It was not until about the beginning of March, when his attention was drawn to Mr. M.'s condition by Mrs. M. and the statement was made with regard to the revolver, that he thought it was necessary to advise a sanitarium. Dr. T. saw him about that time and made an examination. He states he paid particular attention to M.'s physical condition. At the same time he mentions that M. did show some nervous states, but that he did not make a mental examination. I was

and he had made a success of things. I think any idea of this kind showed decided mental deterioration at that time.

Q. What about his will power, Dr. Z.?

Witness: What do you mean by "will power?"

Q. There would be marked enfeeblement of the power of the will, so as to act independently of others, or would he be subject to being influenced by the will of others more than he would in his normal condition?

A. He would be uncertain in undertaking new lines, but in regard to actions that were caused by his delusions, he might be very obstinate. He would, perhaps, be dependent upon the help of other people in undertaking new lines, and new thoughts, and new ideas, and new affairs—he would feel his own enfeeblement.

Q. From the personal examination you made of the patient at the time, could you say whether arteriosclerosis that he had was known as cerebral arteriosclerosis or not?

A. Oh, yes. He had definite evidences of cerebral arteriosclerosis.

Cross-examined by Mr. D. H., of counsel for defendant:

Q. I suppose that before you examined Mr. M. you were told something of his case, were you not?

A. Oh, yes, of his case.

By the Court:

Q. Do I understand you to say that very few people whose minds are affected are free from insomnia?

A. No, my Lord; I did not say that. I say there are very few people suffering from insomnia whose minds do not become affected, more or less of course.

not present during the examination of Dr. S. or the examination of Dr. T., but there is a feeling to the effect that they stated Mr. M. was insane. I cannot think it possible, seeing that he was allowed his full liberty, and allowed to journey down to Dalhousie, and allowed to go around the hotels just as he liked. Dr. T. is one of our leading men in the City of Montreal, and a case of insanity would be treated by him like a case of diphtheria—in a case of diphtheria we naturally take precaution to save the individual and to save the people at large. In insanity we do the same thing. So far as I am concerned I cannot think for a moment that Mr. M. was insane in the months of January, February and March. True, he did speak about being ruined, or ruining his family. Dr. S. tells us about him being worried and stating repeatedly that he was ruined, yet he was known to give two thousand dollars to Mr. M. in April. He did not act like a man who had a delusion—a melancholiac. He certainly had nervous symptoms. He was agitated, depressed and emotional. I might say he was peculiar, but all alienists know that peculiarity in character frequently is present in those cases of people who develop melancholia later on. For these reasons and others I conclude that Mr. M. was a sane man in the months of January, February and March, 1912.

Q. Assuming that Mr. M. had been suffering from melancholia during these two months what would you say as to the possibility of persons with whom he came in contact during that period noticing anything about his condition?

Q. It is one of the initial stages of mental trouble?

A. It may be. It is not always.

Q. A man may have insomnia for a few months, and then right himself?

A. Yes.

Q. But if it be continuous?

A. A man may have insomnia from worry or from pain. You have to take the whole case together. Here is a man suffering from arteriosclerosis, and a very definite worry, beginning to suffer from constant insomnia. That is a very different and very serious symptom.

By Mr. H.:

Q. A man suffering from melancholia is not necessarily at all times incapable of appreciating what he is doing?

A. A man suffering from melancholia with mental deterioration is at all times under the power of his delusions.

Q. But there has to be mental deterioration also?

A. Well, even without mental deterioration, a man who is suffering from melancholia would be influenced by his delusions. Of course there are melancholiacs and melancholiacs. If we stick to the point as you know, this man was suffering from involutional melancholia.

Q. That is when you saw him in May, 1913?

A. Yes; I have no doubt in my mind, from the testimony I have heard here, that in January, 1912, he also had it. I have no doubt he had it then. If we just stick to the point, I am perfectly certain that man was influenced by his delusions constantly from time to time, and he had mental deterioration at that time.

A. I think if a man is suffering from melancholia he could not meet his friends as Mr. M. did without displaying it. He could not meet friends from day to day, sometimes twice a day, and not manifest the symptoms of the disease.

Q. In giving your opinion do you bear in mind the facts (assuming them to be true) that Mr. M. did, on certain occasions, during these months make statements to the effect that he or his family or his son were ruined, or on one occasion he said the best thing would be for the three of them to die together?

A. Yes, I have borne that in mind. I believe a sane man might make a statement like that if he was worried as Mr. M. was by his son's condition and other things. He might very well do these things and not mean anything thereby, as the statements were not repeated time and time again.

Q. Would you consider the fact that G. M. was addicted to the use of alcohol would have any bearing on his making a statement of this kind?

A. I believe it would.

Q. In what way?

A. Well, he might be depressed from the effects of the alcohol.

Q. Does that occur?

A. Frequently.

Q. What opinion can you give as regards Mr. M.'s will power and the liability of undue influence being exerted over him these months?

A. I think his will power was perfectly normal.

Q. In addition to reading the evidence of the different witnesses in the case I understand you were present in court every day?

Q. What are the delusions to which you refer?

A. He had delusions that he was ruined, and that he had ruined his family. He had delusions that he was being persecuted and that he was going to be put in jail.

Re-examined by Mr. S., of counsel for plaintiff:

Q. The opinion you have given us, Dr. Z., is based on the ensemble of the facts which you have learned; it is not based on any one particular fact?

A. It is on the whole thing.

Q. Taken together?

A. Yes.

By the Court:

Q. The opinion you give is that this man was insane from the month of January, 1912?

A. Yes.

Q. And continuously insane from that time forward?

A. Absolutely.

His Lordship: The witness has taken notes and has given you certain opinions based on these notes. He took notes of what he considered important in the evidence of the different witnesses. He says these notes represent what to him was important, from his point of view, in the evidence of these different witnesses, and that he used these notes to base his opinion on. The result of his examination of these notes is that he has no hesitation in stating that the man must have been insane in the month of January, and that he never recovered his sanity. Is that correct?

Witness: Yes, my Lord; that is correct.

A. Every day with the exception of part of one day.

Q. From the commencement?

A. Yes.

Q. So you have also practically heard all the evidence from the witnesses themselves?

A. Yes.

Q. In fact, you have read through all the evidence given in the case in addition to hearing it?

A. I read it through quite a number of times.

Cross-examined by Mr. S., of counsel for plaintiff:

Q. When did you see Mr. M. at the asylum?

A. December 11, 1913.

Q. At that time was he sane or insane?

A. Insane.

Q. How long had he been insane

A. I could not say.

Q. You heard the evidence of the other expert witnesses, Dr. E., Dr. C., Dr. D., and Dr. B., to the effect that this man had been insane, in their opinion, since the month of May of that year?

A. From the evidence I had, it is likely.

Q. You heard the evidence of the other expert witnesses, Dr. E., Dr. C., Dr. D. and Dr. B. to the effect that this man had been insane, in their opinion, since the month of May of that year?

A. Well, I cannot say I did. I cannot recollect that. They may have said it or they may not.

Q. Do you agree with that or not?

A. Well, I don't know. They spoke about the condition of the patient developing in May or June. By that they may have meant that the disease started in May or June.

Q. Can you say whether in your opinion G. M. was sane or insane when he was confined to the asylum?

A. He was certainly insane.

Q. How long had he been insane previous to the 15th of July?

A. Well, it is pretty hard to say. He showed symptoms, as brought out by the evidence, before that. Dr. S. speaks about him losing himself and other things. It looks as if in May and June the disease was present.

Q. So that you are not able to say whether or not he was insane in the month of May?

A. I would not like to swear to it.

Q. But I would like to have your opinion and see whether you agree with these other gentlemen who were examined here and who said that in their opinion M. was insane about the middle of May?

A. I would not like to give a date.

Q. Have you any doubt at all that he was insane previous to July?

A. He certainly was insane before July.

Q. Before the 15th of July?

A. Yes.

Q. I think you have said that a person suffering from melancholia cannot hide his condition or dissimulate his condition for any length of time so as to deceive others. Is that right?

A. That is what I said.

Q. Then, how do you account for the fact that one of the principal witnesses for the defendant saw him on an average of fifteen times out of every month, that is to say, every other day from the beginning of the year down to the 15th of July, and never saw anything of the kind?



A. It may be he did not open his eyes or open his ears sufficiently. I cannot answer for him.

Q. But he says he saw him and conversed with him and talked business with him as frequently as on an average of every other day in the month, down to the time he was put in the asylum, and that he saw nothing unusual about him at all. How do you account for that?

Witness: Might I ask what witness that was?

Counsel: That was the witness McL. Did you read his evidence?

A. I must have. Who is Mr. McL.?

Counsel: Mr. McL. describes himself as an insurance agent.

A. Oh, yes; I remember all about him now.

Q. This witness saw M. right down to the time he went into the asylum, every other day on an average. He had conversations with him every other day, and still noticed nothing unusual about him. How do you account for that?

A. I cannot understand it.

Q. Was he telling the truth?

A. This gentleman must have lacked some quality of observation or as they were frequently meeting at the bar—I don't mean to say this in any bad spirit—they may have been taking some spirits. The only other explanation is that Mr. M. was a record case. I never heard or saw of a person suffering from melancholia who could discuss topics of this kind, as described, and still appear normal.

#### DEPOSITION OF DR. Y.

Examined by Mr. S., of counsel for plaintiff:

Q. Have you made a specialty of certain diseases?

#### DEPOSITION OF DR. B.

Examined by Mr. D. H., of counsel for defendant:

Q. Are you practising your profession in Montreal?

A. Of mental diseases for twenty years.

Q. This implies that you have made special preparatory studies?

A. Yes, sir. I entered the asylum of —, where I spent seven years of practice.

Q. You have been seven years there and afterwards you started to practice?

A. I make a practice of mental and nervous diseases. I am attached to the — Hospital, where I am consultant for all mental and nervous diseases, and for two years I have been attached as alienist for the Recorder's Court, to examine all patients that are to be confined in any asylum, at the expense of the government and the city.

Q. Doctor, regarding your report of the interviews with G. M., the 22d of February, the 28th of February and the 2d of May, will you enumerate what are the observations you made as a physician?

A. He (G. M.) appeared very depressed; talked with difficulty; questions had to be repeated before he would answer. Said he was ruined; that his family was ruined; that he was hunted; that they wanted to put him in prison and torture him. He heard voices. He imagined that there were detectives who were watching him; he heard them talking.

Q. Voices? Not yours; voices other than yours?

A. Other than mine or the employees or other patients in the hospital. These were hallucinations. This delirium of the first interview did not appear to me to be very coherent. It did not appear to be a delirium well organized. It

A. Yes.

Q. As a general practitioner?

A. Yes.

Q. You have also given special attention for some years past to the subject of nervous diseases?

A. I have.

Q. Will you tell us briefly what has been your experience?

A. Well, I studied a year in Europe, paying special attention to nervous diseases.

Q. Did you examine G. M., the father of the plaintiff in this case?

A. I did.

Q. On what date?

A. December 7, 1913.

Q. What did you find his mental condition to be?

A. I found him suffering from melancholia.

Q. Have you been able to form any opinion from the evidence and documents which you have read, and which are filed in this case, as to the mental condition of G. M., and his sanity or insanity during the months of December, 1911, January, February and March, 1912?

A. I believe he was sane.

Q. That is your opinion?

A. That is my opinion.

Q. What, in your opinion, based on the evidence made in this case, was the mental capacity of G. M. to appreciate the nature of the transfer made to the defendant of certain mortgages on or about the 5th of March, 1912, and the transaction by which he endorsed a certain note and receipts for bonds as described in this case to Mrs. M. on February 21, 1912?

A. I believe he could understand what he was doing.

Q. That is your opinion?

appeared to me as if he had already presented at that date fresh intelligence. I could not very easily connect the different delirious ideas.

By the Court:

Q. You say at the first interview?

A. From the first interview he appeared as having had a weak intellect. I came to this conclusion later, especially at the last examination, where he did not remember the length of time he had been in the hospital. He was not accurate as to facts, dates, or anything at all.

Q. What conclusions have you come to yourself?

A. That the sickness has existed a long time.

Q. Would you be able to state approximately how long the patient has been suffering from the sickness which you have observed?

A. The examination alone makes it difficult to give a precise date, but the state that he was in when I saw him gave the impression that he had already been suffering from a weak intellect, and would give one the impression that it had existed for a long time back. To arrive at the beginning of the disease and the weakness in intellect, I think one would have to consider or take into consideration the proof brought by other witnesses, who saw him at different periods of his life.

Q. Exactly; and this brings us to what has been stated by the testimonies which have been given here in court?

A. Yes.

Q. You have heard these testimonies?

A. Yes.

By Mr. L., of counsel for plaintiff:

A. That is my opinion.

Q. From the evidence adduced in this case and from the exhibits filed, would you consider that Mr. G. M. was suffering from progressive and continuous melancholia during these months?

A. No.

Q. Do you consider that if G. M. was suffering from continuous and progressive melancholia at this time this condition would have been noticeable to the various friends whom he met and with whom he talked during the periods as described in the evidence?

A. I believe it would have been. As far as I have seen in the evidence, the people who saw him depressed and with these ideas of ruin and so on saw him in the hospital, where he was in contact with his son, who was very ill and who was believed to be dying, or they were persons who had something to do with the business of his son, which was then in a very bad way, as far as the evidence shows. Of course, this was very depressing. At the same time, and on the same days practically, he would meet other people and appear in an ordinary mental condition.

Q. How many cases of melancholia have you had under treatment?

A. I don't know. I have had quite a few.

Q. How many?

A. I could not recollect.

Q. Surely you can recollect within five, or ten, or twenty, or twenty-five?

A. No.

Q. Did you have one?

A. Yes.

Q. Now, considering the intermittence or non-intermittence of this disease, which you have stated is present, what have you got to say?

A. In the disease G. M. suffers from there is no intermission. The disease is continuous.

Q. If you please, will you give me precisely what are the facts in this proof that bring you to this conclusion, in the proof that you have heard that the malady was of long duration?

A. There is what Dr. S. said who examined G. M. on the 8th of January, I believe, and he declares that Mr. M. had ideas of ruin at that time. He has the proof brought by Dr. T. who examined him a little later, and he declares that at that time he was insane. In January he commenced to want to look for a situation, pretending that he had need to work to support his wife and son, who were at the hospital. In December, from the 1st of December, I believe, he found himself absolutely incapable of regulating the affairs of his son.

Q. Have you had more than one?

A. Yes; I have had more than one. I see some of these cases in connection with the nervous clinic. We do not have the certified cases.

Q. You do not classify yourself as being an alienist, but you classify yourself as being an expert in nervous diseases?

A. We have mental diseases in the incipient stages, too.

Q. The nervous diseases form the greater part of your studies?

A. Yes.

Q. In cases where these patients can maintain their self-control and repress the outward expression of their symptoms do you believe they can maintain this self-control for any length of time?

A. Not for any length of time. I think that especially meeting old friends and talking over old times the delusions would come to the surface very quickly.

Q. Can you tell us how many cases of melancholia you have had under treatment and examination?

A. No, I could not.

Q. You cannot remember the number of cases?

A. No.

Q. Did you have two cases?

A. I have seen far more than two cases. I have seen many cases at the hospital.

Q. But I am not asking you that. I am asking you how many cases you have had under treatment?

A. I have had a certain number of cases in my private practice, and we have had many at the hospital.

Q. I mean cases of melancholia which came under your observation as a physician—under your personal treatment?

## DEPOSITION OF DR. W.

Examined by Mr. C. H. S., of counsel for plaintiff:

Q. Dr. W., are you an alienist?

A. My specialty is mental diseases and their treatment.

Q. For how long have you been such?

A. Ten and a half years.

Q. You occupy a position in the — Asylum?

A. Yes.

Q. What is your position?

A. Assistant Medical Superintendent.

Q. You were there at the time G. M. was admitted to the asylum, as explained by Dr. U.?

A. Yes.

Q. In the meantime, can you state from recollection on whose application he was admitted?

A. I am pretty sure it was Mrs. M., his wife, because her son was sick at the time.

Q. Who are the medical men who made the necessary certificates?

A. Dr. S. and Dr. T.

Q. Did you examine G. M. when he was admitted?

A. I did. I examined him within a few hours of his admission. It might have been the next morning,

A. Yes, I understand.

Q. How many have you had?

A. I have no idea.

By the Court:

Q. You could not say whether it was one hundred or one thousand?

A. It certainly was not one thousand.

Q. Would it be one hundred?

A. We certainly must have had one hundred showing melancholic symptoms.

## DEPOSITION OF DR. C.

Examined by Mr. S. L. D. H., of counsel for defendant:

Q. Have you specialized in any particular branch of your profession?

A. Yes; I have specialized in mental and nervous diseases.

Q. Will you give us briefly your experience in connection with these diseases as a professional man?

A. I spent four years in Europe, in Edinburgh, London, Paris and Vienna chiefly, attending the best hospitals there in this branch of work. I have had a private hospital of my own for the better part of twenty years; so in that way I have had an opportunity of studying and examining nervous conditions. I established a ward in the general hospital for nervous troubles, with the special intention of demonstrating the prevention of insanity.

Q. What class of patients do you treat in this hospital?

A. Nervous patients.

Q. What do you mean by nervous patients?

A. I mean patients suffering from psychical symptoms—in other words, patients suffering from mental symptoms, in the proper sense of



but it was within a few hours of his admission.

Q. Will you please describe to the court the condition in which you found him?

A. I found him very depressed and emotional. He spoke of suicide and claimed to have attempted it. Do you want all these details?

Counsel: Yes; we would like to have them.

A. He had the delusion that detectives were continually on his track. He stated that he had lost several hundred thousand dollars in poor business deals and that he was financially ruined. He was worrying very greatly over his son, who was seriously ill with typhoid at the time of his admission, July 15. His condition was one of marked mental anxiety and mental pain or depression, and I had not the slightest doubt after my first examination that it was a case of melancholia. The case was very evident.

Q. Was he sane or insane?

A. He was insane.

Q. Could you form any opinion as to how long he had been insane?

A. For one thing, on the papers that accompanied him the statement was made. . . .

Mr. G., of counsel for defendant, objects to the witness giving an opinion based upon what other doctors may have told him.

By the Court:

Q. Do you mean the statements of the doctors on the certificates?

A. Yes.

His Lordship: If the witness is going to base his opinion on the history as described by the other doctors, I think it is admissible. At

the word. I do not distinguish between psychical and mental symptoms indicating insanity. I do not take insane cases in my own hospital. My hospital is not for that, but for a previous stage of the condition.

Q. A pre-insane condition?

A. Yes, a pre-insane condition. Of course there are the organic diseases of the brain, and so on, which are part of the work, but, more largely, the pre-insane conditions have been prominent in my work for many years. I devote my time exclusively to this work. I do not do any general work or that kind of thing. I am a specialist in the proper sense of the word, so far as devoting all my time to these nervous and mental troubles is concerned.

Q. Do you have to deal with depressed or melancholic conditions?

A. Yes. I deal with both depressed and melancholic conditions.

Q. I understand you have examined Mr. G. M., the father of the plaintiff in this case?

A. Yes.

Q. At the Verdun Asylum?

A. Yes.

Q. When did you examine him?

A. On December 11, last, 1913.

Q. What was his condition when you examined him?

A. I found him in a condition of involutional melancholia with delusions.

Q. From the evidence, and from the documents filed, did you form any opinion as to the sanity or insanity of Mr. G. M. during the months of December, 1911, January, February and March, 1912?

A. Yes. I believe he was sane.

the same time, I will take your objection, Mr. G., and will allow the evidence under reserve.

Witness: On the papers accompanying Mr. M., or preceding him—the papers of commitment—it was stated by the physicians that mental symptoms had shown themselves in November, 1911, if my memory serves me correctly. From my own observation of the patient at that time, believing him to be a case of melancholia of his age and that type, to the best of my professional knowledge of that disease, I should say the mental symptoms had existed for some time before he came to us. I say that on my own knowledge, not on the history as given to me or as given to us by the examining physicians. I deduce that, from the man's condition, and the type of mental disease from which he suffered, his mental disease had existed for some time before he came to us.

By Mr. S.:

Q. One year?

No answer.

By the Court:

Q. What length of time?

A. Well, I must answer that to the best of my judgment in such cases. I could not state that, because it is impossible to say definitely; at the same time, from my knowledge of other cases, I should say it would have lasted certainly some weeks, possibly months, possibly three or four months.

By Mr. S.:

Q. That is, the condition in which you found him?

A. Yes, based on my own observation of his case as a type of mental disorder.

Q. That is your opinion?

A. That is my opinion.

Q. Basing yourself on the evidence and on the documents filed, what, in your opinion, was the mental capacity of Mr. G. M. to appreciate the nature of the transactions in question in this case?

A. Quite good as far as G. M. was concerned.

Q. Would you consider from the evidence in the case that Mr. G. M. was suffering from confirmed melancholia (that is, I understand a progressive and continuous disease) during these months?

A. I would not.

Q. Will you give us the reasons for the opinion you have expressed in regard to Mr. M.'s mental condition during the months in question?

A. You ask me why I would consider G. M. as being sane at this time. In order to judge of a man's sanity you first have to obtain his normal condition, as near as you can, in order to learn how much he deviates from that normal in whatever acts he may perform. What I think is an important item in regard to it is this: in looking over the evidence we find that G. M. was always subject to certain eccentricities and when crossed, for example, he would curse or swear, walk up and down and mutter to himself. We also learn that he was a practical joker; that he was a pessimist, that he always hesitated to begin business or undertake anything new; that he lacked confidence in his own business ability. These, I think, are a few of his peculiarities as they came out in the evidence so far. They struck me as bearing on the case particularly. He

Q. What, in your opinion, would be the effect of the condition in which you found the patient on his capacity for transacting business?

A. Well, if you put that as a hypothetical question, I should say he would not be in the full possession of his mental faculties. In all probability there would be an inability on his part to conduct his own affairs with the same prudence and judgment as he would before the attack came on or before any sign of mental disease manifested itself.

Q. Was his mental condition due to any physical disorder, as far as you could discover?

A. I think it was due to a combination of physical causes and mental causes. I would put down arteriosclerosis—disease of the arteries—as one cause, acted on by mental worry and grief over the illness of his son and possibly by other worries. I think the underlying cause was long-continued and progressive arteriosclerosis—arterial disease.

Q. Might I ask you a scientific question? Is there a physical cause for every mental disease?

Mr. G., of counsel for defendant, objects to this question as being irrelevant and illegal.

The question is allowed.

A. In certain forms of insanity there is a well-marked physical cause; in others a physical cause is suspected, but it is obscure and not proven to be the cause.

Q. Will you give us a little more detail as to the conduct of the patient after his admission to the hospital?

Mr. G., of counsel for defendant, objects to this question as being irrelevant and illegal.

was always inclined to borrow trouble, as a natural man. These conditions existed in this particular individual for a number of years—for instance at the time when he was a member of Parliament and was doing the business of the country. These were also characteristic conditions in the individual in later years. The next point I consider is the evidence of the doctors who saw him at the time. If we go into this medical evidence we find that Dr. S. examined Mr. M. early in January; that he found certain physical symptoms and certain nervous symptoms. Dr. S. found certain nervous symptoms—he found insomnia; he found inability on the part of the patient to fix his attention. He states that he answered questions slowly but correctly; that he was considerably agitated, and he spoke of ruining his boy. I believe these are all the symptoms Dr. S. mentions as a result of his examination in January. Of course, it is very hard to keep all these things in one's mind where there is such a mass of evidence, but I would like to feel that I had covered all the symptoms mentioned by Dr. S., because, being a medical man in charge of the case, one would naturally expect to place a great deal of weight upon his opinion. Dr. S. says he saw M. from time to time, but he made no further examination and that there were no new symptoms up to the time he left, just prior to March 5. There was no mental alteration in the man that Dr. S. considered sufficient to add to these symptoms at that time. Mrs. M. made a statement that Mr. M. had a revolver or threatened to shoot himself. Dr. S. says distinctly that if she had not

The objection is reserved by the court.

A. The patient continued to show this marked mental depression. He showed insomnia. He was restless. He refused his food frequently, because he stated it was tainted with human fecal matter, put in by his persecutors. At one time, about a month after his admission, I think it was in August—he attempted to suicide by driving a pair of scissors which he clutched suddenly into his right eye. They penetrated several inches and resulted in loss of vision in the right eye. Of course, we watched him very carefully after that, as we considered him then to be dangerously and actively suicidal. From that time on until the present he has shown a deterioration in every way, with a persistence of these depressing delusions of various kinds, and delusions of a persecutory character. For instance, he told me to-day that he was to be taken and exhibited through Philadelphia and Chicago as a terrible example of human guilt; that he was to be made to eat manure on the main street of Montreal. I just mention these to show you that his delusions are of a very horrible, terrifying and depressing character.

Q. What is the condition of G. M. at the present time?

A. His condition is one of involutional melancholia, verging into presenile insanity, and with certain signs of general mental and intellectual deterioration.

Q. Has there been any time since his admission to the hospital in which you would say he was better?

A. Absolutely no.

Q. Has there been any time in which you would call him sane?

made that statement he would not have considered him insane, or advised his going to an asylum. Dr. S., in the earlier stages, did not have any apparent anxiety as to this man's mental condition or as to his being insane, otherwise, he would have suggested a consultation, which is a very natural and ordinary thing to do. There is no evidence of his having done this. He prescribed for M.'s sleeplessness, which he said was the main cause for which he had examined him. I think it is clear that with no change in his mental symptoms, or nothing new in his mental symptoms (which I believe are the exact words used by Dr. S.), there was not sufficient ground in them to consider him insane. We now come to Dr. T.'s evidence: When Dr. T. took the case over from Dr. S. I presume Dr. S. would give him some history of the condition; in other words, would tell him what he had found on previous occasions and what the conditions were, so that Dr. T. would not have to go to the trouble of determining for himself the previous condition of the plaintiff; that he would have a history of the case from Dr. S. This is the usual practice and I think it is a fair inference to say it was done in this case. Dr. T. examined the general condition of the patient. He says he has no recollection of making an examination from a mental point of view. If Dr. S. had said to Dr. T., "This man is insane," surely Dr. T., with his experience in everything and with the practice he has had—if this man was in the position of a confirmed melancholiac—would have considered the mental side of it. However, the only statement Dr. T. makes in re-

A. No.

Q. Is his present condition in accordance with what you would expect from the nature of his disease?

A. Yes; it certainly is.

Q. Is it in accordance with what the symptoms as described by the doctors who gave their medical certificates for his admission would imply?

A. It is exactly what I would look for in melancholic symptoms appearing in a man of his age, with his history of alcoholism plus arteriosclerosis; yes.

Q. What is the effect of such a condition upon the capacity of the patient to transact business?

A. Well, insofar as his mind and emotions are dominated by these delusions, he is quite unable to form the same clear conception and judgment, because his mind fails to have the proper concentration that it had before and his judgment is not normal.

Q. What have you to say about his will power?

A. Well, his power of voluntary action and so on is affected by these delusions, as well as weakened or lessened—decidedly impaired.

Q. Does it make the patient liable to be easily influenced?

Witness: You might elaborate that question a little.

Q. I mean to be led or influenced by others.

A. Along the line of his delusions, possibly yes. In order to protect himself from these supposed persecutors he might be influenced quite easily along such lines, having delusions.

Q. If the patient's will is weakened in consequence of the disease,

gard to it is that he thought it was a mental condition rather than a physical one. He made no diagnosis—he said so distinctly—before the examination in July. It is true M. was depressed when Dr. T. saw him, and that he had a downcast look, and did not reply to Dr. T.'s questions, and spoke of the ruin of his family. This, however, did not impress Dr. T. with the man's insanity, or that he was even suffering from delusions. These were to my mind simply examples of the great discouragement under which the man was at that time, and Dr. T., I think very wisely, says that he cannot state whether that condition was transitory or permanent. As I say, that was the only examination Dr. S. or Dr. T. made prior to the end of April and this examination took place during the first two weeks of March. There is one important point, I think, in Dr. T.'s evidence to which I should refer, he having been there at the time and seeing the case subsequently. He expressed the opinion that the case developed in the months of May and June. That, from the physician in attendance at the time, who saw the patient constantly, was a matter which impressed me in regard to the evidence. Another important point, to my mind, was the number of witnesses of all classes of life who saw Mr. M. during the months in question—that is, December, January, February and March. We find members of Parliament, managers of large businesses, and so on. In fact, people in all walks of life, ladies as well. They are prepared to say, and do state in their evidence, that many of them had known him for a number of years and had



as you have indicated, would it not result necessarily from that weakening of the will power that a person might be led to do, at the suggestion of others, things that he would not do in his normal condition?

Mr. G. objects and the objection is maintained.

Q. Where the mind of a patient is weakened, in consequence of the disease which you have described, what is your experience as to the patient's power of resistance?

Witness: Power of resistance in regard to what?

Counsel: To the will of others?

A. That varies very greatly, dependent upon the form of mental disorder which one encounters.

Q. I mean the form which you have described?

A. In cases of melancholia of that character, while they are under the domination of their delusions more or less, they are very likely to follow out their dictates altogether regardless of anything that is brought to bear on them from an external source, especially if the influence has to do with trying to convince them that their delusions are delusions, because their delusions are really true beliefs to them as far as they go. My experience with cases of melancholia is that while acting under the domination of their delusions they are not easily led by outsiders. The delusions dominate them rather than the outsiders.

By Mr. G.:

Q. They are influenced by their delusions?

A. Yes.

talked with him on a great variety of subjects at various and irregular times throughout the whole interval—not for a week or two weeks at a time, but a day here, and perhaps three or four days later on. In that time they did not find that G. M. was in any way abnormal.

Q. If Mr. G. M. had been suffering from delusional melancholia (which I understand is continuous and progressive) in your opinion would it have been evident to the various persons with whom he came in contact during these months?

A. I think it certainly would have been apparent.

Q. Presuming that Mr. G. M. made a statement to his wife, about the end of February, that he thought the best thing that could happen would be for the three of them to die together (as appears from Mrs. M.'s evidence), would that indicate, in your opinion, that he was insane or suffering from confirmed melancholia at that time?

A. No. It would not; for the simple reason that, as a rule, any person suffering from melancholia—a melancholiac—the delusion would begin gradually, and would be repeated much more frequently. This was an isolated statement to the effect that it would be better for them all to die together. He did not make any attempt or do anything to show that it was a delusion in any sense of the word. It was simply a statement made in a moment of depression.

Q. Assuming that he made a statement to the effect that he had ruined his boy or ruined his family, and that he had ruined himself, dur-

By Mr. S.:

Q. That is, with respect to the subject of their delusions?

A. Surely.

By the Court:

Q. Is the mind of a melancholiac, so far as it governs his ordinary conduct, liable to work along normal lines?

A. Absolutely no. It is not. At no time is his mental condition not influenced and governed to a certain extent in all his intellectual processes—all the processes of thought, emotion, judgment, perception and so on—except by the delusions and the depressed character. That influences him at all times, and consequently you cannot say that a case of melancholia has a normal mental process going on at any time.

By Mr. S.:

Q. Did you hear the evidence given by Dr. T. and Dr. S.?

A. Yes.

Q. In your belief, from the time these gentlemen first examined G. M., considering the condition in which they found him, was there any time since then that he had been normal?

A. To the best of my knowledge and belief and experience with mental cases of that character and of that age, and knowing what I may know of their development and their progress, I should say from the time Dr. T. and Dr. S. found that man as emotionally depressed as they say he was—from that time until the present day he has not been normal mentally.

Cross-examined by Mr. G.:

Q. Do I construe your last answer correctly in saying that you

ing the months of January, February and March, in your opinion, would that indicate that he was insane and incapable of appreciating the nature of the transactions with M. referred to in this case?

A. No. I think not. If G. M. had believed he was ruined he would have shown it in his dress, and he would have shown it in his changed life, just as any other melancholiac would do.

Q. Supposing, for the sake of argument, that G. M. was suffering from a delusion that he was ruined, or that his family was ruined, during the months of January, February and March, what have you to say in regard to his failure to refer to this when speaking to the many people with whom he came in contact?

A. Assuming that he had these delusions, he would have been talking to every one about them. He would have been pounding it into everybody he came across.

Cross-examined by Mr. L., of counsel for plaintiff:

Q. Dr. C., for my own satisfaction and perhaps for the satisfaction of the court, will you kindly tell me what is a medical specialist?

Witness: A mental specialist?

Counsel: No, a medical specialist?

A. One who devotes his entire time to the treatment of a certain condition.

Q. That implies first that a man has made a special study of medicine?

A. Presumably, yes.

Q. And, of course, the narrower the limits of the specialty the better

consider the man ceased to be normal mentally when the delusions began or would you antedate it?

A. I would state that the disease was in the early stage of establishment at the time Dr. S. and Dr. T. found him in that abnormally depressed condition, with these delusions that he had lost everything, and wasted his substance and so on. I would consider the disease was established then.

In rebuttal:

Dr. W., examined by Mr. C. H. S.:

Q. You have already been examined on behalf of the plaintiff in this matter?

A. Yes.

Q. You have heard the testimony which has been given with regard to the possibility of recognizing or determining whether a patient is or is not afflicted with cerebral arteriosclerosis?

A. Yes.

Q. What is your opinion with regard to that?

A. I think it is possible for a doctor to determine with a reasonable degree of assurance whether a case is suffering from cerebral arteriosclerosis or not.

Q. Speaking with regard to the power of a patient suffering from delusional melancholia to conceal his condition, what have you to say to the court?

A. My experience is that patients suffering from melancholia and who have the disease established, may for limited periods conceal their delusions, and may, at certain times of the day—notably in the late afternoon or early evening—really appear brighter and seem to show that the melancholia has lifted

the specialist, other things being equal?

A. Provided it included the whole of that specialty; not half of it.

Q. So that a specialist who would have devoted all his time to the study of mental diseases would, other things being equal, be more competent than a specialist who has distributed his attentions to diseases of the nervous system and diseases of the mind?

A. You ask my opinion about that, and I say emphatically no.

Q. Give us the reason?

A. Well, if I am not correct in my appreciation of your question I would be glad to be put right. From a practical point of view the alienist sees these cases only after they are certified insane. The preceding stage of the condition is certainly more or less unknown to him, and for the very simple reason that these people—

Q. (Interrupting) Which people?

A. The people who are admitted to the asylums. They are there in an advanced stage of the condition. They are declared insane, but probably there is a long period of confirmed mental condition preceding that which the alienist does not see, unless he is also a neurologist and among these cases.

Q. So that a gentleman who like yourself, has devoted his time to the study of nervous diseases and what I would call the semi-ready stages of insanity—

A. (Interrupting) The preliminary stages of insanity—

Q. Is better fitted to pronounce an opinion than the specialist who is known as an alienist?

A. My opinion is that the man who is conversant with these con-

somewhat. I have seen many cases of that kind. I might refer to a case which came under my observation and treatment at the hospital not long ago, as a typical case to prove the point I refer to. We had a case at the institution, a man aged 55 years. He came to us in April and went away in May. During the last fortnight of his stay with us he appeared very well so far as his wife and family were concerned. When they came to see him he appeared to be very well and they objected very much to leaving him there. They took him out against our advice. We said that the man would be better with us and that he certainly was not a well man, but was a danger to himself. Notwithstanding this, they took him away. He did not show any depression to them and he did not talk over his delusions with them. When they came to see him he appeared to be sane. Indeed, he did not talk over his delusions very much with us, but, at the same time, we recognized that he was not very well. They took him out of the hospital on May 13, and about the middle of August he committed suicide. In other words, the disease was still there. It was continuous all the time. There was no remission and there was no intermission. The man was not well. He had the disease and he had these delusions which caused him to think that he should leave this world and that he should take his own life. As I say, he successfully concealed these delusions from his wife and family.

Q. Was he capable of talking rationally during that time?

ditions and who sees them every day would be able to give a better opinion than the man who only sees then when the pronounced stage is reached.

A. He talked very clearly and connectedly on many topics and in regard to many things.

Cross-examined by Mr. A. G.:

Q. Will you please listen to the question which I will read you from page 13 of your deposition given on behalf of the plaintiff in chief?

By the Court:

Q. Is the mind of a melancholic, so far as it governs his ordinary conduct, liable to work along normal lines?

"A. No. Absolutely. It is not. At no time is his mental condition not influenced and governed to a certain extent in all his intellectual processes—all the processes of thought, emotion, perception and so on, except by the delusions and depressed character. They influence him at all times; and consequently you cannot say that a case of melancholia has a normal mental process going on at any time." Is that answer correct?

A. Certainly it is correct. The process is not normal. I did not consider the case of the man I mentioned a moment ago as being normal. What I said was that he appeared so.

Q. But you did not confine yourself in that answer to saying that it was not normal. You went on to say that all the processes of thought, emotion, judgment, perception and so on, are influenced exclusively by the delusions and the depressed character?

A. I still maintain they are.

Q. You maintain that a man may absolutely create the impression he is cured, although every one of the processes of thought, emotion, judgment, perception and so on,



somewhat. I have seen many cases of that kind. I might refer to a case which came under my observation and treatment at the hospital not long ago, as a typical case to prove the point I refer to. We had a case at the institution, a man aged 55 years. He came to us in April and went away in May. During the last fortnight of his stay with us he appeared very well so far as his wife and family were concerned. When they came to see him he appeared to be very well and they objected very much to leaving him there. They took him out against our advice. We said that the man would be better with us and that he certainly was not a well man, but was a danger to himself. Notwithstanding this, they took him away. He did not show any depression to them and he did not talk over his delusions with them. When they came to see him he appeared to be sane. Indeed, he did not talk over his delusions very much with us, but, at the same time, we recognized that he was not very well. They took him out of the hospital on May 13, and about the middle of August he committed suicide. In other words, the disease was still there. It was continuous all the time. There was no remission and there was no intermission. The man was not well. He had the disease and he had these delusions which caused him to think that he should leave this world and that he should take his own life. As I say, he successfully concealed these delusions from his wife and family.

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A. I still maintain they are.

Q. You maintain that a man may absolutely create the impression he is cured, although every one of the processes of thought, emotion, judgment, perception and so on,

are influenced exclusively by these delusions?

A. Not influenced exclusively.

Q. You said in your answer, "At no time is his mental condition not influenced and governed to a certain extent in all his intellectual processes—all the processes of thought, emotion, judgment, perception and so on, except by the delusions and the depressed character?"

A. Yes; that is quite correct.

Q. They are influenced to a certain extent by the delusions. Do you maintain that he can be in that state and influenced at all times in all his intellectual processes by his delusions?

A. To a certain extent, yes. A man may be influenced by his delusions that he is a terrible criminal, for instance, and must get out of the world, or must rid the world of himself; yet, he may be able to conceal that delusion, so as to get an opportunity to kill himself.

Q. You are now suggesting that he may conceal his delusion so as to get an opportunity to carry out his plans?

A. It is merely one activity of the human mind.

Q. Do you suggest that he can carry that out to the extent of absolutely hiding his condition so that he will appear sane?

A. I do not suggest it. I absolutely know it. I have seen it.

Q. How do you know this man did not have a remission?

Witness: Which man?

Counsel: The man you mentioned a few moments ago as an example?

A. Because he was under my personal care and observation every day up to the time he left the hos-

pital. When he went out of the institution he was still suffering from melancholia, and he had these delusions, although, as I say, he did not show them to the very friends and relatives on whom he depended to take him away and who actually did take him away against our advice.

Q. Your idea is that this man was hiding his delusions for the purpose of being taken home?

A. At the time, he was.

Q. That would be the explanation of his behavior?

A. Yes.

Q. Nevertheless, he was not hiding his delusions from you?

A. No.

Q. He was perfectly confident that they were safely hidden if he hid them from his relatives and friends, and told them to you?

A. I could not say as to that.

Q. In any event, you did not think of suggesting to his relatives that they should question him in regard to these delusions?

A. I told his relatives, as I have said two or three times, that I considered the man dangerous. He mentioned these things to me, and I knew they were still in his mind, and that they ought not to take him away.

A. What sort of melancholic was he?

A. He was one of the involutional types of melancholia.

Q. What do you mean by "one of the involutional types?"

A. Depending on retrogressive and degenerative change physically. He was a man about 55 years of age.

Q. What was the cause of his melancholia?

A. I am not absolutely sure, without looking up the records. I think there was some statement made, but I am not sure what it was.

Q. What was his delusion?

A. He had the delusion that he ought to die. He thought he had lived a very sinful life, and it would be much better if he were out of the world. The man was a plasterer and had not been working for some time. He was not able to work, and he thought he was better out of the world.

Q. Do you suggest that these cases of men able to dissimulate completely to a certain class of people, while exhibiting their delusion to another class, are frequent cases, or do you deny the statement which I think I heard made that the general tendency of the melancholic is to harp on his delusions, unless there is some special reason which keeps him away from them?

A. I should say the average case of melancholia does dwell very continuously on the delusions. There is no doubt about that.

Q. What was the age of G. M.?

A. He was said to be 66 at the date of his admission.

Q. You say you can discover whether arteriosclerosis affects the brain or not?

A. I did not say that. I said that a doctor meeting certain symptoms in his patient might very reasonably come to a well-founded conclusion that cerebral arteriosclerosis did exist. That was the gist of my statement.

Q. Of course, you would not pronounce a man insane simply because you found evidence of arteriosclerosis, or even of cerebral arteriosclerosis?



A. No. I certainly would not.

Q. A mental examination would be necessary?

A. Decidedly.

Q. You may have a man suffering from arteriosclerosis and showing some of these symptoms which, according to you, indicate the probability of cerebral arteriosclerosis, and this man may be insane from other causes which have nothing to do with the arteriosclerosis?

A. Certainly.

Q. You have known of such men or have heard of such men having recovered.

A. I will not say that. I know I have had many cases of insanity recover, who have had arteriosclerosis in different parts of the body.

Q. In the present stage of science, the fact that they recover would be a conclusive demonstration that the insanity was not due to arteriosclerosis?

A. Yes.

Q. Take the case of a man who is insane, and who has arteriosclerosis and exhibits the symptoms which, according to you, indicate that he had not cerebral arteriosclerosis?

A. I would say not. If I found these symptoms pointing to arteriosclerosis, I would not expect a man to recover.

Q. That is not the idea I have in mind. I say take a case where the circumstances are such that, in the present state of science, your diagnosis would be that the man was suffering from cerebral arteriosclerosis. In other words, I ask you whether in the present state of science your diagnosis that he would not recover would be infallible?

A. No; nobody's diagnosis can be infallible.

Q. Was the patient to whom you referred as an example suffering from the same form of insanity as G. M. is suffering from?

A. I would call it an involuntional melancholia, but the sclerotic condition was not nearly as marked.

Q. That is to say, the sclerotic condition in M.'s case was more marked?

A. Yes.

Q. Was it the same class of melancholia?

A. Yes; I would class it the same. We diagnosed M. as a case of involuntional melancholia. I would also diagnose this other case as one of involuntional melancholia. Of course, any two cases of involuntional melancholia may show a diversity in certain symptoms.

It is hoped that a perusal of the above evidence will establish beyond doubt the foundation of which this paper is the superstructure, viz:—that there is great need for more agreement in the rendering of evidence by alienists and neurologists, and that such a diversity of findings from known facts as appear herein can only obstruct and not hasten the ends of justice.

NOTE: To Mr. Justice Cross, Mr. Henry J. Elliott, K. C., and Drs. G. Villeneuve, F. E. Devlin, and T. J. W. Burgess, of Montreal, I am indebted for valuable aid in the preparation of this paper. Dr. H. V. Robinson has compiled the expert evidence which was submitted in the case.

## THE MODERN TREATMENT OF INEBRIETY.\*

By IRWIN H. NEFF, M. D., FOXBOROUGH, MASSACHUSETTS.

The modern conception of habitual drunkenness demands that there be developed a practical method of handling such cases. Any system proposed which is put into practice must be sufficiently elastic so as to permit of its universal adoption.

State care is preferable and a centralization of authority is essential. The carrying out of the purposes of any plan should be authorized and controlled by the medical profession. All the details of such a plan, both preventive and curative, should be censured by medical experts before submission to the public for approval and adoption.

Any institution built for the care and treatment of drunkenness should be constructed so as to meet the selective requirements of these people; it should also allow for regional departments directed by the parent institution, which should be considered the administrative head.

In order to be effective, any legislation which is enacted for the amelioration of an evil must be supported and upheld by public opinion. Heretofore, owing to the absence of reliable data and lack of co-operation, the many isolated attempts which have been put forward to solve the problem of drunkenness, have met with little or no success. It has been said with considerable truth that there is much to fear from excess in drinking, but there is also much to fear from excessive statements which experience soon discovers to be unsupported by facts. Recently the correlation of scientific facts and the co-operation of allied interests have produced satisfactory working capital which can readily serve as a basis for the inauguration of a uniform method of combatting this condition. The reasons for the universal demand for a plan which will modify or control habitual drunkenness may be said to be two: first, the sentimental reason, namely, a realization of the fact that the public owes to humanity a debt which is considerably overdue; secondly, the economic reason, namely, a realization that

\* Read at the seventieth annual meeting of the American Medico-Psychological Association, Baltimore, Md., May 26-29, 1914.

chronic drunkenness is a destroyer of efficiency and is so related to poverty, crime, insanity, social decadence and physical disease, that any method advanced for the improvement of conditions will not only check human waste and disease, but will necessarily lessen the expenditures fostered upon a community entailed in the care of the victims of drunkenness and their dependants.

That there is a need for a uniform and practical method for caring for cases of habitual drunkenness is clearly shown by a review of the laws on the statute books relative to the regulation of the liquor interests. The numerous requests for more stringent legislation is additional proof that we realize that we are in need of state laws which will more completely protect humanity from the evils resulting from the abuse of alcohol. A careful review of the many laws enacted in different states for the regulation of the sale of alcoholic beverages convinces us that the great mass of legislation was not proposed for the abolition of a personal privilege, namely, prohibition of what many consider a constitutional right, but for the purpose of controlling drunkenness, a condition coming from the abuse of an agent which is capable of producing intoxication, disease and economic waste. The question before us when we deal with the inebriate is not one of state or world-wide prohibition, but one more clearly of the education of the individual to his relation to a substance deleterious to him; in other words, the relation is that of the physician to the patient. It must be remembered that the following remarks refer to the inebriate and do not refer to the chronic alcoholic or to the persistent social drinker. When we consider the inebriate we must remember that we are dealing with an individual who has a distinctive constitutional peculiarity, in that the habit of drinking is engrafted on a weakness.

R. W. Branthwaite of England clearly expressed this condition when he said that "this constitutional peculiarity may be acquired by long-continued indulgence, but it is more properly inherited as a diathesis in most cases, remaining latent or becoming evident according to circumstances or environment." When this constitutional predisposition or fault or diathesis, or whatever we may call it, is present, it is permanent. Doubtless some people who abstain from the use of alcohol possess it unknowingly, other persons who take alcohol realize its existence and spend their

lives battling against the weakness; some succeed while others fail. If an inebriate becomes a sober individual the peculiarity back of the habit which we call drunkenness still remains a factor to be reckoned with during the whole life of the individual.

Disregarding for our present purposes our personal opinions on "the alcohol question," we must acknowledge the need for a more complete and consistent plan for the bettering of a condition which is one of the nation's greatest handicaps. The medical profession has been singularly lacking in an appreciation of the fact that the problem of the amelioration of this condition is within its province; indeed, it is only within the past few years that sufficient detailed and scientific study has aroused the public to the necessity of co-operating with physicians in an effort to overcome a social evil which is progressively increasing.

Sociologists, philanthropists and jurists have for years struggled with the question and have failed to arrive at any satisfactory solution. The physician has made feeble and half-hearted efforts to substantiate his ideas, and the resulting conflict of opinion has only served to demonstrate the inefficiency of the many methods of caring for the habitual drinker. It must be said to our discredit that the neglect of the medical man to properly appreciate this question is in great part responsible for the futile and many-sided efforts which have been made by different interests to cure or control drunkenness. One of the greatest difficulties which the medical man has to overcome is the need of convincing the public that the many alleged "cures" for drunkenness now existing are not countenanced by the medical profession, but are the product of a successful system of quackery which has flourished profitably for years.

Assured of the co-operation of the public and taxpayer, any state can now proceed, under medical supervision, to develop an organization which will squarely meet this problem, for the study of drunkenness is clearly a medical-social study which should be supervised and controlled by the medical profession.

For the past six years Massachusetts has given considerable study to the problem of drunkenness. During this time two special commissions have prepared and submitted legislative reports, which have resulted in legislation enabling the commonwealth to make a substantial start in putting into practice the



recommendations made by these commissions. An analysis of the several reports shows many things in common and demonstrates particularly that any plan inaugurated by the state for the solution of the problem should be approached from two vantage points. Any system proposed must be met from a preventive and curative side by the adoption of a uniform plan with centralized control. Both reports referred to are emphatic in declaring that the method which has existed from early times of committing persons to penal institutions for drunkenness is wrong in principle and should be abandoned. Moreover the commissioners agree as to the need of state non-punitive control for the care and treatment of habitual drunkenness.

The treatment and care of the inebriate does not call for institutional treatment alone, but demands a more widespread policy which includes a consideration of the personal equation and opportunity for developing a system which will allow for differentiation, segregation and individualization. We can therefore consider the proper handling of this class of individuals from two points of view; first, the institutional department; secondly, the non-institutional or out-patient department.

Recognizing that the inebriate requires specialized care and treatment, an institution built for inebriates must be built around the individual, and must be so constructed as to administer to the varied types of inebriety and also allow for the development of the different departments. Briefly expressed the requirements for the proper constructive treatment of the inebriate are as follows:

- (A) Sufficient land for agricultural development.
- (B) Sufficient land for industrial training and work shops.
- (C) An opportunity for segregation of the types.

To meet these requirements a large tract of land, certainly not less than one thousand acres, should be available. This land should be adaptable, lending itself readily to outdoor employment and diversion for the patients. Massachusetts has committed herself to the colony system of caring for these people, the cottages being grouped in colonies. A number of these cottages have already been built and are occupied by patients. In order to permit of segregation and individualization these groups of cottages or colonies are suitably situated and distributed over the large

tract of land. The size of the cottages, the unit of the system, is governed by the type of case to be cared for in each colony. The maximum number accommodated in any cottage is twenty-five. In addition to these colonies with their respective homes and farms, a group of buildings has been erected which serves as an administrative and receiving group. These service buildings are conveniently located so as to permit of an economic management. Further extension of the hospital will consist of additional cottages which will be built at the different colonies. The colonies, briefly expressed, are as follows:

- (1) A colony for incipient, or hopeful, inebriate cases.
- (2) A colony for more advanced male cases, supposedly men who are in need of custodial care.
- (3) A colony for refractory male cases; men who do not lend themselves to ordinary methods of treatment, and who require more or less restraint.
- (4) A colony for inebriate women.

It is at once apparent that the essential part of such a grouping is the cottage or unit, which must be so situated and constructed that it will readily lend itself to the needs of the patient groups.

#### NON-INSTITUTIONAL OR OUT-PATIENT DEPARTMENT.

The need of this department is evident and can be met by the establishment of an out-patient office which should be situated in a metropolitan area and not too remote from the parent institution. This department is an integral part of the hospital. Its purpose may be defined as follows:

- (a) A preliminary examination of the prospective patient for the purpose of differentiation.
- (b) Visits to patients while at the hospital.
- (c) Visits to the homes of patients before their discharge from the hospital.
- (d) Visits to patients after their discharge from the hospital.

This department is in charge of a physician; home visitations and the vocational bureau are supervised by agents who are responsible to the departmental physician. The department is conducted by the state as a permanent central office, which serves as a center for all out-patient work.

The scheme as developed by the Massachusetts system and which

is now in part in active operation can be graphically presented as follows:

(1) A state hospital for inebriates developed on the colony plan, with a sufficiently ample and flexible equipment for the different types and grades of cases of habitual drunkenness.

(2) An out-patient department, with broad and well-defined duties.

(3) Detention hospitals serving as adjunct institutions to the central hospital. These hospitals are to be situated in the cities and towns of the commonwealth. The hospital need not be especially built for the purpose, but should have special features for the care and treatment of cases of acute alcoholism. Briefly defined, the purposes of these hospitals would be as follows:

(a) For the treatment of delirium tremens.

(b) To serve as an observation and receiving ward for the parent hospital.

(c) To provide a clinic for incipient cases of inebriety.

(d) To serve as sub-offices for the out-patient department of the main hospital.

(e) To provide medical officers to visit prisons to examine cases arrested for drunkenness and to determine their fitness for treatment at the hospital.

It must be acknowledged that Massachusetts, with its excellent probation system co-operating with the hospital and its state farm allowing for the detention of the criminal inebriate, is well equipped for the state care of drunkenness as above outlined.

The proposition as above described is elastic and can be modified so that its purposes can be carried out by any state or municipality. All the departments above enumerated should be under uniform management, with trained medical specialists and centralized control. The essential feature of this method is that the patients realize that they are receiving consistent treatment afforded by the hospital, and that they respond to it in ways that they do not respond to the diversified management of the asylum, jails, reformatories and almshouses under which in succession they now pass.

It is believed that the system as above outlined affords distinct advantages for the solving of the complex problems associated with drunkenness. There can be no doubt that the adoption of

such a method under state control would not only result in economies to the state, but would contribute to the sciences of medicine, psychology and sociology new and important data on the problem of drunkenness.

I feel that this brief description of the plan to which Massachusetts has committed herself would be incomplete if I omitted a description of our method of medical treatment of inebriates.

Hospital treatment for male inebriates has been provided by Massachusetts at the Foxborough State Hospital for twenty-two years. Until recently all cases were committed from municipal, district or police courts (except in Suffolk and Nantucket counties, where they are committed from probate courts) upon certification of two physicians that the man is "subject to dipsomania or inebriety either in public or in private or . . . is so addicted to the intemperate use of narcotics or stimulants as to have lost the power of self-control," and "is not of bad repute or of bad character apart from (his) habits of intemperance." Since the reorganization of the hospital in 1907 provision has also been made for the admission of voluntary cases (Acts of 1909, Chapter 504) either directly or from the criminal courts, as one of the terms of probation. During the year ending November 30, 1913, the number of cases received by commitment was 171; the number of voluntary cases received was 577.

The curative methods of dealing with inebriety now employed at the Foxborough State Hospital consist in special individual treatment to build up the body, mind and character of each patient. A detailed physical examination is made of each person received, and all diseases or defects which are noted receive appropriate medical treatment. The bodily health is built up by means of nourishing food, out-of-door work and by supervised exercises. Good physical health is the foundation upon which cure of habitual drunkenness must be built. It is, however, only one of the conditions of cure. Work and rest are so arranged as to habituate the patient to regularity, for inebriates are characteristically persons of irregular habits, and the creation of order in their lives serves to reduce temptation and to provide the basis of moral living.

Study of the mental condition of the patient supplements the physical examination, and the causes of his inebriety, both indi-

vidual and social, are sought. Successful curative treatment of inebriety is not medical in any narrow sense, but is mental. There is no known specific. "Cures" are accomplished through suggestion, not by drugs. A type of work is chosen for each patient which will be congenial to him and will prepare him to enter some steady employment upon discharge. Latent abilities and interests of the patient are sought out and developed in so far as possible, not only in his work, but in his leisure hours.

The essential element of hospital treatment of inebriety is, however, not the building of healthy bodies and regular habits; not the industrial or other training, for the well-conducted prison can furnish these. The hospital alone, of all state institutions, can provide those elements of suggestion and moral suasion which constitute a large part of psychotherapy. The co-operation of the patient is an essential condition of success in treatment by mental suggestion; this co-operation cannot readily be elicited in the prison where discipline and restraint tend to arouse an attitude of antagonism or resentment on the part of the inmates towards officials. Clearly those patients who come to the hospital voluntarily for cure (whether they come directly or are sent by a judge or probation officer as one of the terms of probation, or procure their own commitment under Chapter 504, Acts of 1909) are more likely to co-operate and to be benefitted than are persons who are committed to the hospital against their will. The physician, by means of repeated suggestion made under favorable conditions, persuades the patient of the danger to himself and to others of his habit of intoxication; of the necessity of complete abstinence from the use of all liquors; of the success that can be achieved through careful work and regular habits. Special suggestion is made to each patient on the basis of his peculiarities, to quell his special weaknesses, to develop his ambition, re-develop his self-respect and supply new interests. No two cases are precisely alike, nor can they be successfully treated by any stereotyped plan. Diagnosis by a physician of specialized training in nervous and mental diseases, and continuous suggestive treatment under his direction, adapted at every point to the physical and mental needs of the patient, are essential to cure.

The curative treatment of the patient is continued after his discharge from the hospital through the out-patient department.



Prior to the establishment of this department in 1909 the discharged patient often had to return to an environment that was unfavorable to his new resolve of abstinence, to unemployment, to drinking friends, or to a comfortless home. Discouragement from these sources, if unrelieved, will in some cases reduce health and, through counter suggestion, overturn the treatment of the hospital. To tide the patient over this crucial transition period the Foxborough State Hospital has appointed a special out-patient physician, who becomes acquainted with each case while it is at the hospital and with the patient's family. Before the discharge the family is shown how they can co-operate in perfecting the cure. Work is found for the patient before his release. He is associated with local persons or social clubs or religious organizations that will look after him and provide temperate friends and wholesome amusement. By frequent visits to the hospital and visits from the out-patient physician the suggestion made at the hospital is reiterated until years of continuous abstinence prove that further oversight is no longer needed.



## THE PREVENTION OF SUICIDE.\*

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The safe-guarding of those in custody on account of mental unsoundness is a problem for their guardians, which will not be considered here. The problem we have to study is one of preventive medicine, and concerns thousands of suicides due to distress of mind, the result of psycho-sociological conditions, before which, to judge by their great increase, society shows a helplessness which, in view of present psychopathological knowledge, is reprehensible.

For example, to a young man or woman the object of his or her affection fails or ceases to reciprocate. Under this cross, bitter pessimism as to the future dominates the mind, and forthwith the only solution which is seen is grasped and life terminated. Or the situation may be such as that portrayed in *La Giaconda*, where jealousy complicates the situation. Or the business or professional man finds the struggle too great, and feeling he cannot endure the implicit reproaches of wife and family, or the disregard of associates, he flees. He may fly to drink, or crime; but, as we may learn from the newspapers, death is a frequent recourse.

Now, not everyone subjected to psychical traumata of this and kindred nature takes refuge in death. In the struggle of motive, the conservative features may preponderate, or may even at the last moment during attempt at suicide, reassert themselves, as in the case of Benvenuto Cellini or in that of the more modern and better analysed instance of the young Genevese lady reported by Flournoy.

### CASE I. A REMARKABLE EXAMPLE OF SELF-COMMUNION BY DREAM HALLUCINATION, PREVENTING SUICIDE.

A young married woman, much admired and petted, became acquainted with a young doctor of Geneva while at a summer resort with her mother.

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His seriousness and force of character excited an admiration and respect in the spoilt child, who had always before been treated rather as a puppet than as a thinking being.

Although she was of agnostic tendencies, his firm faith, maintained without rancor, only increased her respect during their numerous discussions on philosophy and morals.

Having suffered the serious chagrin of an estrangement from her husband and the belief that she had contracted a serious disease from him, she became preoccupied by the thought of suicide, and this culminated one night before a ball after one of her children had wept on being repulsed by her on account of her fear of infecting the child.

Returning home tired after intentionally showing unusual gaiety at the ball, the horror of her lot seized her so strongly that she impulsively rushed to the wharf on the lake alongside which she lived, and bent over to look in the water, which at the moment attracted her strongly, when suddenly, from the water, rose a vivid picture of young Dr. T., looking stern, and talking in the harsh voice he had once used to her in reproof. He clasped her round the waist and by the shoulder, and led her home, all the while pointing out the enormity of her contemplated act, and showing her how her duty lay with her children.

As to what dictated Mdme. A.'s choice of the person who appeared in the hallucination, even if telepathy were proved true, an occultist explanation can be excluded here, because Dr. T. did not even know that she dreamt of destroying herself and he had no particular thought of her at the time. Moreover, for three months they had been corresponding, being led to do so without deliberate intent; and indeed at that moment Mdme. A. carried inside her corsage a letter received just before the ball, of which she had postponed the reading until a more tranquil moment. Hence an undertrain of thought of him must have run through the evening, especially as she felt that Dr. T.'s opinion about her disease and course was the most valuable she knew; he was the incarnation of prudence, reason, morality, trust in the ideal. Even her annoyance at his opinions came from her feeling of their superiority. He had taken the place of a director of her conscience. For even although, unlike a hypnotiser, he had never directly commanded or suggested her in any way, his whole relation was one huge suggestion towards courage and hope.

As in most dreams, a certain fantasticalness showed itself. Thus the doctor was dressed in Alpine costume; this was due to the preponderance of a first impression (for the first time she saw him he was thus dressed). As a matter of fact, he had never placed his arm round her waist nor caused her to kneel at his feet, nor reprimanded her directly, nor spoken to her in the second person singular, although they always spoke in French.

Although three and a half years have elapsed, no similar phenomenon has occurred to Mdme. A. At first she believed in its reality, though hardly in its miraculousness, but after a few days she suspected that it was illusory.

The evocation of Mdme. A.'s doctor friend in hallucination is comparable to the association of one's hypnotiser with the idea he has suggested. There is no need to insist upon the usefulness of Mdme. A.'s experience; it saved her life. There is no need to invoke a metaphysical explanation, dramatic as was the event; for it was merely the sudden ascendancy of motives of superior force, which had been temporarily buried in the mind, during the fixing of her attention upon her misfortune, and during the emotional upset when she repulsed her child.

Benvenuto Cellini relates a similar experience which occurred during his imprisonment by the Pope. His careful preparation for suicide was suddenly interrupted by an invisible presence, which violently threw him into the corner of his cell; whereupon the idea of self-destruction disappeared forever.

Both these experiences are of *oniric* character, and have the same psychological effect as would occur from a heart to heart conversation in which the frank avowal of our troubles dispels the morbid notions which we have tried to hide, but which nevertheless eat at the heart like a canker.

But not every one has within himself a powerful director of conscience ready to spring to the rescue when danger threatens. So these people require assistance to overcome their psychological disorder. It must be remembered that the appeals to common-sense and religious motives have failed in the persons who commit suicide; and I regret to say that there are many instances in which medical advice sought for fails to assist. This is not because medical science has not learnt to cope with such a malady, but because the doctor consulted is too seldom conversant with psycho-



pathology, and so takes refuge in a vague tradition which leads to the giving of bromides, strychnine, douches, rest-cures (the name provokes derision), advice to change scene, or suggestive or would-be cheerful reassurances based on what the physician believes to be common-sense. What is required of the physician is to realize that the patient is in need of the kind of sense which is as yet "uncommon"; and all these procedures fail in effect, because they are mere shots in the dark, not even entitled to the term empirical; for they are not based on successful experience nor on sound induction nor reasoning; they are merely imitative therapeutics applied without proper diagnosis of the patient's need, and without adequate comprehension even of the effects they produce, and entirely without reference to the process at work in the patient.

What then should be done? Simply what is done in all real medicine: (1) To understand the physiology of the perversion of function presenting itself; (2) rectify the disorder if one can.

Suicide generally denotes disturbance of an individual's power of social adjustment. For example:

- (1) Disease frankly in the body, such as cardiac distress.
- (2) Misinterpretation of reality, as in alcoholic raptus.
- (3) Emotional grief, as love-sickness, bereavement, business failure, etc.
- (4) Simply a wrong idea.

It is the last two varieties which are studied in this article. Concrete examples where suicide was prevented form the best introduction to a comprehension of the principles upon which depends prophylaxis.

#### CASE II. ATTEMPTED SUICIDE; PSYCHOGENESIS, THERAPEUSIS.

A farmer's son of 22, after some weeks of moody behavior, threw himself into a creek. He was quickly rescued by his brother, who reproached him severely. This did not deter him; for a few weeks later he swallowed laudanum. This led to his removal to a sanatorium where, after a few weeks, he crushed and swallowed an electric light globe. Later, he gained access to a medicine cupboard, and again swallowed laudanum. So his friends in despair brought him to a doctor friend in Washington, who immediately asked me to see him.

Examination showed no physical disorder; but I discovered that there existed a serious psychological situation, which no one had even suspected, much less attempted to penetrate.

The boy was so ashamed of himself, although still determined to commit suicide, that it was hard from his whispered utterances to reveal the facts from the analysis of which was furnished the very simple explanation of his distressing predicament. To state the position briefly: Upon this boy had devolved, since the death of his father, the management of his mother's farm. But a younger brother had succeeded in interfering a good deal with our patient's plans, much to his mortification; and when also neighbors' meddling was acquiesced in by his mother the situation became intolerable, as he had already failed in an attempt to work happily in another environment which he tried for over a year. So that suicide seemed the only escape.

The manner in which the psychological situation was ascertained is best judged from a transcription of the questions and answers of part of the examination:

"What is the matter?"

"Stomach troubles; if I could get well I would be alright."

"Have you any pain?"

"No."

"Why are you then complaining?"

"Because my bowels do not work."

"Why take so much laudanum?"

"Because I think I should be better off if dead." To a further question. "If I could be cured I would be content." "I could not stand being worried by my brother of 19, and my sister who is 24, and my mother. I want to go and work for myself; I should get on better." (He had forgotten to mention his sister; and when she was mentioned, he stammered.) "They pick on me; for example, if I get up too early; and I always feel I could not do the things I want to do. But when I went to California, I felt uneasy, even when working alone. I have been dissatisfied all my life. I do not know what my trouble is or what I have done different from any one else." To a further question, "I went to school."

"Have you done anything with which to reproach yourself?"

"No. I think there must be something wrong with my brain." To a further question, "The whole case is imagination."

"Why do you think so?"

"I do not know."

"Since when have you thought so?"

"Since four years ago, when neighbors would interfere with what I had done on the farm; for example, in planting the corn, people would comment upon it, and my mother would take their advice and overrule my way."

"Why do you take it so hard?"

"Because I have poor judgment."

The inquiry was then pushed with regard to his relations with the opposite sex. He declared that he had liked their society, although he did not dance and was not "immoral" as he called it; but he confessed his bashfulness and also that he thought girls were not worth spending so much money upon as was necessary; he did not think they were dependable, and he had decided not to marry because of seeing so much of married life; he had never cared for any particular girl, although he had often desired them, but had not the "face" to make advances towards what he thought to be wrong, as at school boys and girls had been separated; besides the girls laughed at his timidity. Accordingly, he told the other boys that their indecent talk was wrong; and was laughed at for his pains and made still more bashful and ashamed.

However, he had dreamed of erotic situations, which made him feel ill; and he feared it would injure his health. As a small child, his dreams had been terrifying, such as falling and being killed, or being run away with by horses; but these had not troubled him since. There had been no spontaneous diurnal emissions; but he had provoked them until he was 18, and had then ceased to do so, as other boys often teased him about it, and said that he would be impotent, as he had ruined himself; hence he was very much ashamed.

*Interpretation.*—The failure of this boy to stand up for himself was due to his own shame at the onanism he had practised and his fear that it was injuring his mentality; so that he was not able to stand up against other boys, by whom he was much teased; in consequence of which he withdrew from social life, especially where girls were concerned, and became taciturn and irritable.

He had to confess that if he could be well of what he thought incurable, viz: "a hopeless mental inferiority which masturbation

must have caused," he would be willing to live, and would like to work.

*Treatment.*—He was assured, and examples were given him to show that he was quite mistaken about the effects of onanising; and he was asked to think over until the next day the explanation I gave him concerning the genesis of his shame and timidity, meanwhile promising not to attempt suicide until he had seen me again.

The next day discussion was resumed; until in less than a week the boy could be trusted alone, not only in the hospital grounds, but in town. He went home in ten days perfectly cured, and has been at work and in good spirits ever since, now nine months ago.

The treatment was conducted in a general hospital; and the maximum of freedom was allowed the patient from the first, the greatest tact being urged upon those who nursed him.

#### CASE III. LOVE SICKNESS IN A MAN.

Another case of contemplated suicide, in a young banker, was caused by a period of prolonged strain and overwork, culminating in a serious rupture of an engagement which had lasted six years. The patient was lachrymose, agitated, trembling by fits and starts; he would rush from the table suddenly with the desire to kill himself, or break into tears without provocation, especially when with his family. At his work he appeared comparatively calm, but it was only by an intense effort, which further debilitated him. He had lost forty pounds in weight. His relatives exhorted him to "buck up," "forget it," or sometimes chaffed him about it all. This only aggravated his distress, which a progressive insomnia kept augmenting.

The treatment used was to convince him of the need of distraction from his painful ruminations, and that this could be done only by hard physical work which would at the same time increase his resistance to painful memories, by removing the weak irritability of his nervous system. He was sent to the country two or three times before the right kind of place was found, and before he learned to arouse himself from the bodily lethargy and mental concentration upon his troubles. Eventually the right place was found, where wood-chopping and farm work kept his mind oc-

cupied and restored him physically. He now feels better than he has for ten years, and is again at the head of his business.

The love sickness of this patient was only one of the factors in the case, but it is frequently the outstanding feature of a nervous breakdown. It is very wrong to meet it lightly. The proper analysis of the situation should always be undertaken and the co-operation of the patient enlisted towards overcoming the troubled mental state. There is practically always some psychological fact concerning which the patient needs enlightenment.

#### CASE IV. CURE OF CHRONIC FEAR.

A lawyer, 28 years old, gradually withdrew himself from the society of friends, later denying himself to all but one. He abandoned work and began to neglect food. At night he would pace the floor for hours. He looked haunted and ashamed. He twice took steps towards suicide. There is no need to enlarge upon a picture so familiar. Suffice it to say he is cured. He was most distrustful of the possibility of cure, as he had six months previously visited specialists who had failed to benefit him. As he described it, their procedure seemed to have been somewhat crude attempts at hypnosis with suggestive assertions denying his symptoms and their cause, which he had declared to be a state of fear.

It was mainly in the presence of other people that his fear came over him; and he was much ashamed all the time because of this fear. It was quite different from the timidity of adolescence. As a small boy he was noted for his bravery, and would fight against the boys of the neighborhood.

The cause of his fear was unknown to him; and he believed it was hereditary, as one of his brothers was worse than himself and had become a wanderer whose whereabouts would be unknown for months at a time. The patient had been fighting against this fear at least since his college days; he had tried playing football to make him courageous, but without effect; and so when he graduated he plunged into a camp of rough lumber-men and took his part as a laborer with the rest. Six months of this gave him still greater admiration for courage, but in nowise improved his own. He then returned to civilization and plunged into his studies and office work, hoping to attenuate the fear which gripped him; but instead



of this he gradually lost mastery, and after six years of struggle fell into the state in which he came to me.

*Genesis.*—After a physical examination, which disclosed no important features, except great loss of weight and a high degree of erythema, psychological exploration was begun by my stating to him that either he was, as he believed, a physical degenerate or there was some psychological cause for his fear; in which latter case the discovery of that cause might lead to the finding of a means for its removal and the ending of his fear. He was then told to search his memory for fear-bearing experiences in early life, but could think of none. Then period by period running back from his college days had attention turned upon it; until the patient recollected to have been morbidly fearful at each time; until finally he declared that he had always been afraid. He was then asked what incidents of his early childhood had particularly frightened him, and at first recollected nothing. Wild animals, darkness, fire and people were each in turn presented as possible factors. But it was not until the remembrance of a near relative was recalled that the key of the situation was found. It seemed that this individual's ideal of up-bringing was the hardening process, and that the theory he held was that every boy's moral welfare required the knowledge of fear. These two objects were combined in such a procedure as throwing the lads into the water while they were unable to swim, to fish them out only when they were going down almost breathless. In winter, a favorite method was to throw the boys while asleep in the morning into a bank of snow and snowball them home to the door. Another procedure was to chase the children with a stockwhip from the front door to a tree in the distance. The result of all this was not hardening, but a breeding of chronic fear in these two lads. The patient's recollection of these performances reached back to the age of 4. But he had completely put out of his mind these incidents, and indeed failed to take into consideration his cowardice as a young boy, believing it to have originated in the high-school.

*Treatment.*—When the source of the fear was discovered, the patient declared that he did not see how this knowledge would benefit him. It was then explained to him that his fear was merely a physical habit and not an instinctive reaction.

He was told that habits can be reformed if intelligent effort is employed, but that he was in no condition to begin reformation of habit until he had slept and eaten regularly for some days. When he objected that he had long since given up narcotics, as he was worse than before taking them, he was told that I never found it necessary to give narcotics; that I should induce sleep without them, and that after this he would be less unwilling to eat.

Accordingly, treatment was begun by my visiting him in bed and hypnotising him into sleep. He slept eighteen hours, and then carried out the dinner program we had previously arranged. Hypnosis was performed three times in all, but not on consecutive nights.

In the meanwhile re-education was begun. To make a long story short, this consisted merely of a reconstruction of the fear situation of his infancy, and the pointing out of the non-necessity of the fear sequence which had occurred, and the insistence upon the possibility of reconstruction of his reactions towards himself and the world. Numerous instances of the dependence of emotion upon idea\* were given; and he was instructed concerning reconditioning the reflexes as investigated by Pawlow and Crile; and he was shown the physiological perniciousness of the fear impulse.

He struggled with the situation bravely; but I left him alone after what proved too short a period, namely, four days; and he lost courage and began to relapse, until a friend drew my attention to the situation after a week. We then resumed relations, as he felt the need of help. After four more days of re-education, the tide turned and he obtained control of his fear. He celebrated the occasion by an impressionist account of his situation, from which I extract what follows:

"I've won! I've licked him! I've driven away the beast that was driving me mad. As soon as I knew just what he was, and why he came, I poked him with my finger, and he busted. He's not gone entirely; he's crouched growling nearby, waiting to jump on me again. And occasionally he gives me a twinge, such as some men get when passing a looking glass. I laugh at it. I'm on my back no longer; I'm fighting—I'm fighting now. And my

\*"Role of Affectivity and Intellect in Traumatic Hysteria." Williams, in *Journal of Abnormal Psychology*, June, 1910.

battle's all but won. I wrote my last letter on Friday. Yesterday I had fun. I got up singing in the morning, dressed carefully and went down town. I ate my breakfast slowly, but made the waiter scurry. I roamed the streets. A week ago I slunk into a restaurant, because I was fearfully hungry, unshaven, unshorn, and unkempt, and the waiters all laughed at me, and I hurriedly gobbled my food, and crept trembling out again. I went back there yesterday and bullied the whole crowd. One of them came up grinning, and I looked him in the eye, and the grin changed to a smirk. I kept him standing waiting, while I read the menu through. And I said, 'Bring me this and this and that, and Waitah, hurry! and don't you dare to not to do so always.' Ten days ago I sneaked up to the Sherman statue, by moonlight, and looked at the statue of a soldier, longingly, and wondered how he could be. Yesterday I walked up to him laughing, and wished I could shake his hand."

*Reaction.*—It is over a year now since the above account was written and the patient is now successfully practising his profession, and is still happy, not to say buoyant. At first indeed he was so expansive that I suspected a periodic psychosis, in which my intervention was a mere coincidence; but that that is not the case seems to be shown by the gradual subsidence of the extravagant behavior which the patient at first showed. Besides, another inattention wherein no such doubt could arise. It was that of a woman of 28, whose vision was restored by removal of congenital cataract. Dr. Reid Russell of Asheville, the operator, informed me that the patient's reaction was almost maniacal in her joy at her new sensations and at her unaccustomedness to the adaptations they required. So I interpret this young lawyer's extravagance of behavior to his incapacity at first to adjust himself to the new manner of looking upon the people who surrounded him, his former ever-present dread having been displaced by a disregard almost contemptuous with a consequent effervescence of the ego disconcerting to those who previously knew him.

*Interpretation.*— This case is an instance of:

- (1) An anxiety state induced by mechanism other than that postulated as essential by some psychoanalysts.
- (2) The induction of an emotional state directly from an idea.

(3) The forgetting of the initial circumstances which induced the concept which governed the life so detrimentally.

(4) The revelation of the initial circumstances by an analysis so elementary as to be no more than a particularly intelligent anamnesis, in that it neutralized scepticisms and antagonisms and proceeded with patience.

(5) The failure of catharsis per se to alleviate the condition.

\* (6) The need of re-education, that is, psychological reconditioning, for the remaking of the mechanism.

#### THE ETIOLOGY AND PREVENTION OF SUICIDE.

But not all maladjustments lead to suicide.

(1) Some persons lack the courage to die.

(2) Some lack the practical initiative, or the power to act; are aboulie.

(3) Some believe it wicked. So not all painful and seemingly hopeless distresses bring suicide.

(4) Changes in physical states aggravate distress and diminish resistance. But all is psychological in mechanism, that is, the act is dependent on the stored impressions of memory and their associations into painful complexes, which pervert the will to live.

Even the less reflective and more automatic mechanisms are psychological in type, that is, they are modifiable (conditionable as Pawlow calls it), by appropriate stimuli.

The act of suicide, then, is psychological, a perversion of the instinctive will to life and power, by means of a conditioning of that impulse reflex into its opposite by stimuli we may call pathogenetic.

It is not a matter of physical defect of brain or body at all. It is merely the unfortunate utilization of the impressionability which constitutes human superiority towards an accidental course, which happens not to make for conservation.

It is like a turning of the wheel towards the precipice in order to avoid obstacles in the road which appear insurmountable.

The Pawlow dog which cannot escape the idea of the whip shown him, manifests his terror by ceasing gastric flow.

\* The hypnosis used was merely incidental to secure sleep upon certain occasions.

The suicide who cannot escape the idea of the horror of existence manifests it by the automatic reflex of jumping through the window to escape imaginary enemies, as in alcoholic raptus, or by the more complex ratiocinations of the acutely disappointed or the chronically obsessed.

The *remedy*, where the Pawlow dog is concerned, is to recondition the perverted gastric reflex, by bringing other ideas into association with the whip. The remedy, where human beings are concerned, is just the same in principle, but of course more complex in practice; but our means of altering the associations of human beings are vastly more rich than in the case of animals, thanks to the resources of human speech and the influence it brings. They constitute psychotherapy.





THE PRESENT STATUS OF THE APPLICATION OF  
THE ABDERHALDEN DIALYSIS METHOD  
TO PSYCHIATRY.\*

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Direct injection of a foreign proteid into the blood stream gives rise to the formation of a substance (proteolysin) capable of breaking down that particular proteid into simpler products. If, for instance, egg albumin be injected into an animal, there is later found in the blood stream a ferment capable of destroying egg albumin by catalysis. Similarly, albumins derived from a given organ, when injected into another animal, will give rise to ferments of specific action. Still further, Abderhalden claims that a given cell proteid—*e. g.*, liver-cell albumin—is foreign to the blood stream of the individual and if, through any error in the formation or activity or destruction of this liver-cell albumin, it finds its way into the blood stream, it gives rise here also to ferments destined for the destruction of this albumin, to render it innocuous or to prepare it for excretion. These ferments Abderhalden calls "defense ferments" (*Schutz-fermente*), or better, perhaps, "protective enzymes" (*abwehr-fermente*). The specificity of ferments formed against foreign proteids (bacterioly-sins, proteolysins) may be said to have been proven. Abderhalden claims that ferments formed against the cell albumins or their products native to the individual but foreign to the blood stream are also absolutely specific, and this forms the crux of the theory. Frank, Rosenthal and Biberstein, in a general study of this question, conclude that there may occur in the circulation proteid ferments of an exquisite specificity, and others of non-specific nature, and that excessive flooding of the circulation with foreign proteid seems to generate non-specific ferments.

In certain disorders of the metabolic processes connected with cell-nutrition and cell-activity, unaltered cell proteids or

\*Read at the seventieth annual meeting of the American Medico-Psychological Association at Baltimore, May 26-29, 1914.

incompletely or falsely catabolized cell proteids may find their way into the blood stream, where they act as foreign materials. This is the condition known as "disfunction," in the sense of Abderhalden, and the presence of these catabolites \* may give rise to the formation of specific anti-ferments.

Abderhalden's method for determining the presence of these ferments rests on the fact that the proteid molecule is too large to allow of diffusion by dialysis through an animal membrane, while certain products of its catabolism by proteolytic ferments—i. e., the peptones—are diffusible and may be recognized by a chemical reaction. Thus, if we place inside a thimble of such membrane an albumin recovered from a given organ—e. g., brain and serum from a patient in whose circulation there occurs an anti-brain ferment—the action of the ferment on the albumin results in the formation of peptones which diffuse and can be recognized in the liquid surrounding the thimble by the biuret or ninhydrin tests.

The first practical application of this was to the diagnosis of pregnancy. The theory on which this is based is that the cells of the chorionic villi or their proteid constituents or catabolites find their way into the maternal blood stream and there stimulate the formation of anti-ferments of specific type. When blood serum containing such anti-ferments is brought into a dialyzer with proteid extract of placenta, disintegration takes place, with the formation of dialyzable substances which diffuse and are found outside the thimble. Normal sera (in the sense of sera of non-pregnant individuals) are found to be lacking in ferments which will split placental proteid and hence give a negative reaction. I need not here go into the results of the test as applied to pregnancy.

Fausser was the first to apply the tests to cases of mental disease; his first report appeared late in 1912, and since then the literature has grown to a considerable amount. Before taking up the results of these investigations, it may be well to call attention to the greater difficulty encountered here in obtaining the various

\* The term catabolites is here used to convey the meaning of *Abbauprodukte*. It may be remarked that this term could hardly be considered to cover unaltered cell proteids, but it suggests the group of split products resulting from incomplete or faulty catabolism.

organ albumins than in the pregnancy test. It is an easy matter to obtain fresh placental tissue for the preparation of the proteid, but the finding of material suitable for extracts to test for the presence of ferments against the other organs of the body is more difficult. Perfectly fresh material from cases of accidental death is of course the ideal, but anyone who has undertaken the collection of such material will appreciate the difficulties. Usually a considerable number of hours elapse before such a cadaver finds its way to the autopsy table, and the possibility that autolytic changes may have taken place during this period must be considered. In general, the freshest autopsy material comes from hospital cases where death has been anticipated; and here, of course, one is dealing, in theory at least, with pathological material, and as yet we have no definite knowledge as to how far ante-mortem disease conditions may affect the reactions. Lampe and Papazolu have demonstrated that in cases of Basedow's disease the reactions are much stronger when proteid extract of a thyroid from a case of Basedow's disease is employed. One or two investigators have followed this lead in using extracts of brain and other organs for testing similar cases—*e. g.*, general paretic brain extracts against serum from cases of general paralysis, etc.—but so far with no noteworthy variations in the results obtained by the use of extracts from other human sources. This difficulty in obtaining proper extracts has naturally suggested the use of animal organs. Abderhalden has experimented with this idea and reports good results. Kafka similarly found them useful, but this phase of the subject is still in a very early experimental stage.

In Fauser's first paper he reported positive reactions against thyroid in all five cases of frank thyreogenous disease, and in one severe case also a reaction against brain substance. In a series of cases of dementia præcox he found five positive reactions against testicle extract in eight cases, and eight reactions against brain in eleven cases. In six cases of tabes and general paralysis he reports reactions to brain substance in all. Later, in another series, seven male dementia præcox cases gave positive reactions against testicle, and four female against ovary. Six cases of this series also gave reactions against brain.

Following this publication, came a considerable number of

papers which for the most part supported Fauser, but also showed a considerable number of dementia præcox cases in which the thyroid gland or thyroid gland plus brain, with or without the sex glands, was positive; and Fauser, in 1913, in reviewing the subject, interprets the results as follows: For the majority of cases of dementia præcox (Fauser's own cases were chiefly hebephrenic), there is a primary disturbance—*i. e.*, a "disfunction"—of the metabolism of the sex glands. For a small minority a disfunction of the thyroid also appears. An hereditary disturbance is suggested for the sex glands, and an acquired for the thyreogenous. Fauser suggests as a theory that the blood foreign albumins characteristic of the sex glands, when thrown into the blood stream either unaltered or incompletely or improperly catabolised, stimulate in the blood the specific ferments, and, either by themselves or as split products from their catabolism by the ferments, work damage on the brain, thereby giving rise to a disfunction of the brain, which in turn results in an anti-brain ferment. In the small minority the reactions do not appear, or come and go, in some instances suggesting a parallelism with the clinical course. In end stages also the reactions are sometimes negative; *i. e.*, the disease is no longer an advancing process, but a defect stage.

In cerebral syphilis and general paralysis, reactions are found chiefly against brain substance, though occasionally also against other organs.

Fauser claims reactions against sex glands, or sex glands plus brain or thyroid, or thyroid plus brain, or sex glands plus thyroid plus brain, to be characteristic of dementia præcox, and offers such reactions as a means of differential diagnosis from manic-depressive cases, and as a means of indicating the efficacy of therapeutic measures.

Wegener, Fischer, Neue, Theobald, Römer, Bundschuh and Römer, Mayer and others reported series substantially corroborating Fauser's work. Several pointed out, however, that reactions against other organs than brain were met in general paralysis, and that these might follow Fauser's sex-gland-thyroid-brain formula for dementia præcox.

Wegener found in epileptics a brain reaction only, and only then when dementia had set in. Binswanger studied a short series



of epileptics and found 60 per cent positive to brain, and offered the following results and suggestions: Positive reactions speak with considerable surety for epilepsy and against hysteria. During the interval, the cases fall into a positive and a negative group. It is not possible thereby to separate the organic from the "dynamic constitutional" types, as both positive and negative groups appear in each type. A negative reaction in a given case may indicate that there is no progressive anatomical process, and we may hope that dementia will not ensue. We cannot, however, foresee possible increase in severity or number of attacks and the later development of a positive reaction and dementia. Leri and Vurpas studied 25 cases of epilepsy in the Bicêtre and Salpêtrière, and found the same percentage of positive reactions against brain as Binswanger. Here, however, their agreement ended. They could find no relation to the time of the last or approaching attack, nor to frequency of attacks, nor to duration of the disease. Positive reactions were in somewhat higher proportion in the demented cases, but the series is too small for definite conclusions.

Allers studied a short series of cases of various types, using brain only for the tests; he got results less clear than Fauser's, and believes it probable that an unspecific ferment may be present in those cases giving positive reactions to several organs. Willige reported a negative result toward brain in an undoubted general paralytic. Of two brain tumors, one reacted positively toward brain, the other negative toward brain and positive toward thyroid; and he concludes that more observations are necessary before the method can be considered trustworthy.

Mayer points out that while an overbearing majority of the functional and normal cases are negative and the positive reactions in dementia præcox are in the large majority, there are cases where decision is impossible; *e. g.*, general paralysis giving a dementia præcox reaction, or dementia præcox sera with a positive brain reaction only. Kafka reports one case of positive reaction to brain in an acute maniacal excitement, and suggests the possibility of a temporary metabolic disturbance in the manic-depressive at the height of the attack. Golla reported a series with a considerable proportion of the so-called paradoxical reactions. Twenty-five per cent of his manic-depressive cases and 14 per cent of his epileptics reacted to the sex glands. Like-

wise, Plaut found positive reactions to the sex glands in manic-depressive and hysterical cases. Maass reports that imbeciles in certain cases give a reaction of sex glands and brain entirely similar to the dementia præcox formula, while certain idiots give a reaction to brain and thyroid not unlike Fauser's thyroid group of dementia præcox. These reports, if substantiated, are of interest either as indicating that in some idiots and imbeciles there is a continuing state of perverted metabolism rather than a stationary defect, or as suggesting that some of these cases may belong to the class emphasized by the French writers, of an early abortive dementia præcox.

Kafka calls attention to the fact that disturbances of the glands of internal secretion of sufficient severity to result in complete loss of function can give no ferment reaction, and reports negative findings in six cases of non-menstruating idiots and one psychopathic patient who had castrated himself some years previously.

In explaining these discrepancies, the bulk of the blame is laid to errors in technique, and all authors agree that the most painstaking methods are necessary for accuracy. Kafka suggests that no reports should be received in evidence without the publication of the technique adopted, including methods of control, source of organ extracts, etc. Abderhalden himself calls attention to the many sources of possible error of a strictly technical sort, and warns against the use of sera which are not absolutely fresh and against experiments which are not controlled at all possible points. The difficulty in obtaining normal organs sufficiently early post mortem has already been mentioned. It is necessary also that the material be rendered blood-free; and in many organs—such, for example, as the liver—this process is a difficult task. Blood albumins remaining in the extract may becloud the reaction.

One possible source of error, which seems so far to have been overlooked, is the difficulty of getting a strictly pure organ-proteid extract. With the brain, for instance, the process consists essentially in mincing and washing to render the organ blood-free, and then in the removal of the fats by extraction with ether or carbon tetrachloride, and finally in repeated boiling until the water yields a negative ninhydrin reaction for peptones. Taking for granted that the mincing and washing may result in absolute exsanguination, that the extraction of fats may be complete, and that all dif-

fusible proteids may be removed by the boiling, we still have in the organ extract the proteid constituents of the glia cells, of the cell bodies of the connective tissue of the perivascular spaces, and of the endothelial cells and muscle cells of the blood vascular network, any of which, theoretically at least, might prove a source of error in positive reactions.

Still further, many organs, especially those of internal secretion, contain tissues of decidedly difficult structural type and presumably also of functional difference. So far, for instance, no results have been reported where differentiation has been attempted between the anterior and posterior lobes of the pituitary. Pancreas extracts must be considered as containing albumins characteristic of the cells governing both the internal and external secretions. Similarly, testicle extracts must contain material from the cells of procreative type as well as that from the interstitial cells of Leydig.

To review the situation, then, we find that many defense enzymes are reported in cases of organic psychoses which are not found in normal controls, or in any number in the strictly functional psychoses. In the great majority of dementia præcox cases the ferments reported are those against brain or the sex glands, or the two combined. When sex-gland reactions occur, they follow a strict sex-specificity; *i. e.*, male sera react to testicular extracts, but not to ovarian, and *vice versa*. In a smaller number of cases of dementia præcox, thyroid disfunction, either by itself or associated with brain and sex gland, is recorded. Cases do occur, however, in considerable numbers in which these so-called characteristic reactions are lacking; and the same reactions have been found in other psychoses, notably general paralysis and, by one investigator, in idiots. This in itself is, I think, sufficient to preclude considering the test at present as of great value as a diagnostic means until further refinements of technique or larger series of carefully studied cases have been reported. The application at present to border-line cases, for the purpose of diagnosis or the altering of clinical diagnosis on the ground of serological findings, seems unjustifiable, as the clinical study of the case must as yet be considered the means of control of the specificity of the reaction. Further studies should include, also, better classification of cases than simply dementia præcox, and preferably short ab-

stracts of the clinical features of the case. The results of one or two investigators seem to indicate that variations may occur in different stages of the disease; *i. e.*, negative reactions in terminal cases and differences between the various types.

Even if we accept the theory and the results of the most hopeful investigators, we are only brought to the beginning of a wider field of investigation, as by the interpretation of the theory the results speak only for a faulty metabolism in specific organs and as yet give no light on the underlying causes; *i. e.*, the fact that the metabolism of the testicle and brain are disturbed gives no insight into the cause of such disturbance.

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## A CASE OF CHRONIC PROGRESSIVE CHOREA WITH ANATOMICAL STUDY.

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Notwithstanding the copious literature which has accumulated during recent years regarding the affection, chronic degenerative chorea is still quite an obscure disease. This is especially true in respect to its etiology and pathology.

In reference to the etiology of the disease little is known, and, although homogeneous heredity appears to be a pertinent factor in many instances, it fails to explain the origin of the disease in the first diseased ancestor. Typical cases have been reported in which the absence of heredity could be established. It is therefore evident that other factors are to be considered, and, in consequence, some writers, Schabad, Westphal and others, have assumed the possibility of its connection with pregnancy, shock, migraine, etc.

Careful studies in the pathology of this disease are of great interest and importance, as the determination of its morbid anatomy will assist in elucidating the pathology of other diseases associated with choreiform features. It would therefore appear expedient that all cases of chronic progressive chorea should be thoroughly studied clinically, and, whenever possible, pathological investigations made.

Following is a report of the history and pathological findings in a case of Huntington's chorea, being one of three cases of this disease in which the writer has made pathological studies during the past year.

J. W., aged 33, was admitted to the Government Hospital for Insane on August 25, 1909, and died April 1, 1913.

*Family History.*—Father and mother were natives of England. Father died at the age of 87 years, was of good habits, and at no time showed any evidence of nervous or mental affection. Mother died at the age of 78 years, as a result of chronic gastritis. She had always enjoyed good health up to the time of her final illness, and was not regarded as in any way nervous. The patient was the fourth child of a family of six children,



consisting of three girls and three boys. All of his sisters and brothers are still living and in good health. Special care was taken to ascertain whether any indication of the disease occurred in his antecedents, but no such instance could be determined.

*Personal History.*—Patient was born in England, and when an infant was brought to this country by his parents, who settled in New York. With the exception of measles at the age of ten years, his childhood and boyhood were normal. He entered school at the age of seven years and continued through one year of high school, showing a good scholastic record. At the age of 17 years he left school and began the study of music under a cornetist. Subsequently he enlisted in the army as a first class musician, and served eight years. He at no time indulged in the use of alcohol or drugs, and never suffered from any venereal diseases. While serving in the army he contracted dysentery, and was ill for about two months. He was married when 23 years old; his wife had no children or any miscarriages.

*History of Present Illness.*—In the spring of 1905, about four years before the patient entered the hospital, he suffered with symptoms of nervousness. A few months later involuntary muscular movements became manifest, causing his fingers to move and his hands to start; at the same time his arms became affected. About a month later his legs began to show some shakiness. At this stage of the disease the movements were temporarily controllable on voluntary effort.

The jerking of his legs became so pronounced that his gait simulated that of a drunken man, and, before his wife appreciated the nature of the disease, she frequently accused him of being intoxicated. Upon consulting a physician, he was informed that he had chronic chorea.

The involuntary motions increased in severity, and in consequence he was forced to give up his vocation entirely. He made application for a pension, which was granted. In the course of the next few months his facial muscles became involved and his speech became thickened and drawling. The disease pursued a steady, progressive course, and, while the bodily nutrition was well maintained, the involuntary movements were more intense, and about eighteen months later mental symptoms became apparent. He subsequently applied for admission to the National Soldiers' Home, and was admitted May 15, 1909. While at the Soldiers' Home he became very untidy and slovenly, and exceedingly inadvertent in his habits. He was captious, unreasonable, had outbursts of hyperirritability, and finally displayed such distinct symptoms of mental deterioration that he was transferred to the Government Hospital for Insane in August, 1909.

*Condition on Admission.*—The patient was a medium-sized, fairly well-nourished man. His height was 5 feet 7 inches, weight 126 pounds. The skin and cutaneous membranes were normal. No stigmata of degeneration could be detected. Examination of the respiratory system showed no signs of pulmonary disease. The great pectoral muscles were occasionally jerked violently, but this did not embarrass the respiratory movements. Pulmonary sounds were normal, and the heart outlines within normal

limits. The peripheral vessels showed no evidence of sclerosis. The genitourinary system was normal. Temperature and pulse normal. Urinalysis negative.

*Neurological Examination.*—Cranial nerves: The pupils were equal, regular, medium in size, and reacted normally to accommodation and light, directly and consensually. Vision was normal. No involuntary ocular movements were apparent. There was no nystagmus. The corneal reflexes were active and the facial sensibility was not defective. The facial expression was modified by slow, involuntary muscular contractions. No dysphagia was evident. The palatal reflex was normal. Taste, smell and hearing were normal.

Speech was thickened and drawling, the enunciation indistinct and many words went together with a slurring effect.

*Sensory System.*—Tactile sense showed no deviation from the normal. Pain sense normal. He readily recognized marked variations in temperature, but finer changes with difficulty. The muscle sense was intact. Stereognostic sense not impaired.

*Reflexes.*—The arm jerk was equally brisk in both arms. Wrist jerk sharp. Knee jerks were markedly exaggerated. Tendo-achilles were present and equal. There was no clonus. Both the right and left plantar responses were flexor. Cremasteric, epigastric and abdominal reflexes were somewhat hyperactive. The function of the organic sphincters was not disturbed.

*Motor System.*—The musculature, especially of his extremities, was firm and well developed from constant, though involuntary, use. No fibrillation could be detected and contractures were absent. The case presented the characteristic involuntary choreiform movements. During the waking hours they were never absent, but subsided during sleep. With excitement or exertion they became more violent. The muscles of his extremities and head jerk irregularly, and when he attempted to arise from the sitting position his legs were thrown forward with an excessive amount of movement, and in consequence he had difficulty in gaining his equilibrium. A similar exaggeration of his usual involuntary motions occurred when arising from the prone position, and his arms and legs swung about in an aimless manner. When standing his body swayed considerably. In walking his head was generally inclined forward; his steps were about the usual length, but uncertain, owing to the jerky movements, and one leg was frequently thrown in front of the other, causing the gait to closely resemble that of drunkenness. The movements were of about equal intensity in the muscle groups of the legs, arms, head and face. The movements of the facial muscles frequently produced strange contortions and grimaces. The head was jerked forward, backward and laterally, and the resulting peculiar gestures, poses and exaggerations of action were prominent. Movements of the muscles of the trunk also occurred, especially of the abdominal group. He was able by strong effort to somewhat inhibit these movements for a brief period, but this temporary inhibition was invariably followed by a period during which the move-

ments were excessive. There was no evidence of fatigue as a result of the continuous muscular action.

*Mental Condition.*—A general mental deterioration of a slow, progressive character occurred. He was usually inclined to be somewhat retiring and content, although he frequently asked for a parole or permission to return to the Soldiers' Home. His personal habits were slovenly and he required close supervision, owing to his general neglect and inadvertence. A narrowing of the mental horizon and interest and a lack of insight into his condition were perceptible. Occasionally he displayed considerable irritability, at which times he would assault other patients.

There was some evidence of hallucinations and delusions in the early stage of the disease, which, however, were not prominent and faded as the dementia progressed.

Orientation was imperfect. General memory fair, but specific memory for recent and remote events was quite poor.

The physical deterioration of the patient became quite pronounced during the last three months of his life. On the evening of March 25 his temperature rose to 102, and râles could be heard in various parts of the chest. On March 28 areas of dullness were perceptible. The temperature fluctuated between 101° and 103°, and the patient died on April 1, 1913, after a duration of the disease for about eight years.

*SUMMARY OF THE CLINICAL HISTORY.*—A young man 25 years of age, in whose family no hereditary factors could be discovered; whose mental and physical development was up to the normal standard, and who had never had any particular illness, other than tropical dysentery, suffered with symptoms of nervousness in the spring of 1905. In the course of a few months considerable alteration in his general condition occurred. Involuntary choreiform muscular movements developed, first affecting his upper extremities, causing his fingers to move and his hands to start. Shortly after his legs became affected. The involuntary movements of his legs caused his gait to assume a markedly swaying character. Upon consulting a neurologist he was apprised of the true nature of his ailment.

The symptoms steadily progressed; a few months later the muscles of his face became involved, and at the same time articulation became somewhat defective. The involuntary movements became more intense and in about eighteen months disturbance of the intellect was perceptible.

He was admitted to the Soldiers' Home in May, 1909, and while there displayed hyperirritability, became untidy in his habits, and was transferred to the Government Hospital for the Insane in August, 1909.

Examination showed that the pupil reflexes were normal; vision was normal; there was no nystagmus; pain sense was normal; slight variations in temperature were not readily appreciated. Muscle and stereognostic sense normal; tendon reflexes were exaggerated; no ankle clonus or sphincter impairment. It is therefore apparent that the usual signs of organic disease of the pyramidal system were absent, but the involuntary muscular movements indicated a profound disturbance of motility.

The course of the disease was marked by an increase in the motor symptoms and a general mental and physical deterioration of a slow, progressive character. He died on April 1, 1913, from bronchopneumonia.

*AUTOPSY.—External Examination.*—Body: Medium sized and emaciated. Slight post mortem lividity was present.

*Internal Examination.*—Head: The calvarium was symmetrical, wall thick, diploe compact. Dura slightly thickened, but neither adherent nor discolored. Inner surface smooth. Pia stripped readily and appeared normal. The subarachnoid spaces were not distended, and the cerebrospinal fluid was not increased.

Brain: Weight, 1290 gm. The cerebral hemispheres were well developed and showed no visible atrophy or widening of the fissures. The arteries at the base presented no perceptible abnormality. The spinal cord macroscopically appeared to be normal. Transverse sections at different levels were of good size, and no evidence of degeneration was discernible. The membranes showed no turbidity or thickening. The brain and spinal cord, together with portions of peripheral nerves, were placed in 10 per cent formalin for preliminary hardening.

Thorax: Pericardium normal. Heart, weight, 230 gm. Right auricle normal. Tricuspid orifice admits four fingers; valve normal. Right ventricular wall thinned. Pulmonary valve normal. Left auricle enlarged; mitral orifice admits two fingers; valve thickened. Left ventricular wall somewhat thinned. Aortic leaflets slightly thickened.

Lungs: Weight, right, 790 gm; left, 730 gm. Congested and contained consolidated areas in lower lobes.

Abdomen: Liver, weight, 1230 gm. Surface smooth, capsule somewhat thickened. Passive congestion. Gall bladder normal. Spleen, weight, 110 gm. Capsule thickened; surface smooth; pulp



soft and red in color. Pancreas, stomach and intestines normal.

Genitourinary Tract: Kidneys, weight, right, 120 gm.; left 150 gm. Right kidney surface smooth; capsule strips readily; cortex slightly thickened and well defined. Left kidney same condition. Bladder, mucous membrane congested.

The suprarenal bodies, pituitary and thymus glands were removed and examined, but presented no deviation from the normal.

#### MICROSCOPICAL STUDY.

After hardening in formal, the central parts of the cerebral hemispheres, including the basal ganglia, were cut into a series of thin slices, the left by vertical and the right by horizontal incisions. Some of these were utilized for different tissue stains, while others were placed in Muller's solution for the Weigert method.

On the right side, however, the central part of the hemisphere was examined in almost serial sections by Weigert's stain.

Numerous pieces were also taken from various regions of the brain for microscopical study, together with portions of the spinal cord and peripheral nerves.

The following histological methods were employed: Thionin, Nissl, toluidin blue, cresyl violet, Van Gieson, hæmatoxylin-eosin, polychrome methylene blue, Mallory, Mann, gentian violet, Weigert's resorcin-fuchsin, Herxheimer, Soudan III, Bielschowsky, Alzheimer's for glia, Weigert's for neuroglia fibers, Marchi, Spielmeier, Weigert-Pal, Weigert's myelin-sheath and others.

*Meninges.*—The pia-arachnoid showed a very slight thickening, but no infiltration or other pathological changes could be found.

*Brain.*—A study of numerous sections from the precentral gyre revealed some very interesting and important facts. The most striking feature, from a cytological aspect, was the remarkable preservation of the giant cells of Betz. These cells, for the most part, were not altered in staining reaction or morphological characteristics. A few of them only showed beginning pigmentary degeneration with some tigrolysis. Pathological changes of varying degrees of severity were discernible, however, in many of the neurons other than Betz cells, but especially in the deeper layers of the cortex. A more or less extensive deficiency of the chromophilous substance was visible in the degenerated ganglion cells.



In those neurons which showed total tigrolysis a row of small irregular-shaped granules, representing the remains of disintegrated Nissl corpuscles, usually defined the periphery of the cell bodies. In some instances the disintegration was of such a character that the chromophilic substance appeared as a fine powder, while occasionally cells were seen which had lost all their tigroid bodies. Such cells were often reduced to mere shadows. Sometimes the disintegrated granules occurred in groups, and here and there large irregular masses were seen distributed in the cell bodies. Not infrequently ganglion cells were observed in which the Nissl corpuscles presented a normal disposition within the cell bodies, but within the dendritic processes an extremely fine granular disintegration was visible. It was apparent that these changes represented different degrees of the same regressive process.

The nuclei also showed some interesting deviations from the normal. The nucleus was frequently dislocated and occupied an eccentric position near the periphery of the cells. The nuclear membrane was usually distinct, but the contour of the nucleus irregular. Folding of the nuclear membrane sometimes occurred. The nucleus of some cells stained deeply with toluidin blue, but in other instances were swollen, stained lightly and the outline of the nuclear membrane was almost indistinguishable. The lipid substance in many cells appeared to be abnormally increased, and in a few of them an advanced stage of pigmentary degeneration was discernible. This material appeared as irregular-shaped masses of a yellowish tinge lying within the violet-colored cytoplasm.

The most frequent alteration encountered in those cells which showed degenerative changes was characterized by a general shrinkage of the achromatic substance. The chromophilic bodies within the cytoplasm and dendritic processes appeared to be more darkly stained. In a few instances the shrinkage was quite excessive, and in consequence the cells presented a rather elongated appearance. The nuclei in most of these cells were centrally situated and no marked pathological changes could be detected. When stained with the Bielschowsky method for neurofibrils these elements were usually seen to be continuous through the body and processes. The functional activity of such cells was obviously impaired, but there was no reason to assume that it was entirely lost.

Some cells were swollen and the chromatic substance faintly stained. The nuclei were enlarged, pale and of an irregular contour. The nucleoli were likewise distended, and stained metachromatically. The nuclear membrane was oftentimes scarcely discernible and the nucleoplasm presented a stippled aspect. The cytoplasm appeared to have undergone a fine granular disintegration, and the dendritic processes and axis cylinder were traceable for only a limited distance. A few ganglion cells were evident which were distinguished by the intense dark staining of the cell bodies and process. The apical processes were unusually tortuous and could be traced for a considerable distance. The neuraxis process was shrunken. The nuclei were sometimes eccentrically located and the nucleoli increased in size. These cells were deeply stained by the Bielschowsky method. The nuclei were angular and of a dark-brown color. In some, groups of small granules were seen lying within the nucleus.

Concomitant with these alterations of the nerve cells, pathological changes in the neuroglia tissue were evident. These were distinguished by both progressive and regressive changes, an increase of neuroglia cells and a hyperplasia of glia fibers were discernible, chiefly confined to the lower layers of the cortex, where the degeneration of the ganglion cells was greatest. A marked proliferation of small astrocyte cells having densely stained nuclei, and more or less erratic-shaped darkly colored cell bodies, was in evidence, and formed the most distinctive feature of the gliagenetic process. Sometimes the nuclei of the glia cells were distorted, and presented an angular, shrunken, darkly stained homogeneous aspect. The cytoplasm in some instances contained cystic-like masses of lipid substance, which usually stained a yellowish green color. Similar masses of fatty material were occasionally seen in the protoplasmic processes.

Neuroglia cells were also encountered with enlarged, lightly stained nuclei. The nuclear membrane appeared somewhat attenuated. Within the nucleoplasm several metachromatically stained nucleoli were usually discernible, lying within the delicate net-like structure joining the blue stained chromatin particles. The protoplasm was as a rule abundant, more or less ill-defined, faintly stained, and not infrequently contained lipid granules.

A satellitosis was readily perceptible in the vicinity of ganglion

cells, and gliogenous cells were occasionally seen within the cytoplasm and processes of degenerated nerve cells.

With Weigert's method for neuroglia a considerable increase in glia fibers was perceptible in the lower layers of the cortex. The *membrana glia superficialis* appeared normal. The fibers of the cortex were investigated by the Weigert-Pal and Bielschowsky methods. The tangential fibers were in general slightly diminished and in some areas appeared to be extremely reduced. The fibers forming the interradiary and superradiary plexus were likewise somewhat defective.

The majority of the vessels were unaltered and showed no perceptible thickening of their walls or enlargement of the Virchow-Robin space. In the layers of the cortex, however, where cellular degeneration was in evidence, the adventitial spaces of many vessels were dilated, and, as a rule, filled with *Körnchen* cells and irregular shaped masses of greenish-colored lipoid pigment. The nuclei of the adventitial cells were frequently shrunken, stained darkly and their cytoplasm contained granules of lipoid and basophilic metachromatic substance. Yellowish colored granules were also occasionally discernible in the cells of the muscular coat.

The pathological alterations in the postcentral and paracentral convolution were quite similar to those found in the precentral gyre, both in regard to the cellular changes and the fiber content.

An examination of sections from the gyre of the frontal lobe showed diverse alterations in the shape, size and staining reaction of some of the nerve cells throughout the cortex in this region. A few of the cells appeared swollen and faintly stained. In others chromatolytic changes were in evidence. A marked change in the distribution of the tigroid bodies was discernible, and instead of the usual appearance, fine granules were seen arranged in groups or irregularly scattered through the cell. Some of the cells showed a considerable increase of lipoid pigment. A number of cells were encountered which appeared shrunken. Eccentricity of the nucleus sometimes occurred. In the markedly altered cells the nucleus was sometimes shrunken and difficult to distinguish from the protoplasm of the cell, owing to the fact that it stained uniformly throughout. A disintegration of the neurofibrils was visible in some instances by the Bielschowsky method, and the tangential fibers were more scanty than usual. The pathological

changes affecting the glia cells resembled those described in the precentral region. Proliferation of the neuroglia cells occurred mostly in the lower layers of the cortex. The nuclei appeared shrunken, excessively stained and frequently presented a homogeneous aspect. Glia cells were occasionally encountered in the vicinity of degenerated ganglion cells, having rod-like nuclei. Regressive changes were present, and accumulations of pigment were discernible within the protoplasm.

The adventitial spaces of some of the vessels were distended and contained products of disintegration, but otherwise appeared unimpaired.

The cellular alterations in the cortex of the insular region, the temporal and occipital lobes, were analogous to those found in the frontal gyri, but somewhat less extensive.

In the hippocampal region some of the pyramidal cells were shrunken, elongated and deeply stained. No marked proliferation of the glia was present, or pathological alterations of the vessels.

*Basal Ganglia.*—In the optic thalamus circumscribed groups of ganglion cells were encountered which presented a striking appearance, owing to their pale staining in contrast to the surrounding cells. Many of the ganglion cells showed intense pathological changes. In some of the cells the chromatin granules appeared as a fine dust in the center of the cytoplasm and in the vicinity of the nucleus, while at the borders the granules were larger and more darkly stained. Most of these cells had become round in form and their processes showed changes similar to those occurring in the cell body. The nucleus was, in many instances, pressed to the periphery of the cells, stained more darkly than usual, and was sometimes difficult to distinguish from the cytoplasm. Some cells were swollen and the chromophilic bodies appeared as a lightly stained fine dust; the processes were likewise palely stained. Although the nuclei of these cells were distorted and eccentric, the nuclear membrane remained intact. Regressive changes in the nucleolus were frequently discernible. These alterations were readily distinguishable from those types of cellular degeneration in which nothing remained of the cell body other than a row of irregular shaped basophilic granules surrounding enlarged metachromatically stained nuclei whose nuclear membrane had disappeared. Sometimes the nuclei presented an elongated or oval form and



were more deeply stained than the granular masses; in other instances the centers of the nuclei had vanished.

Cells with a reticular arrangement of the chromatin and with more or less shrinkage of the nuclei were not infrequently encountered.

With the Bielschowsky method the neurofibrils in many cells were seen to be discontinuous and appeared as fine granules. Increase in the neuroglia tissue was quite evident. Glia cells having two or three nuclei were often encountered, and areas were present where the neuroglia elements grouped together in large numbers.

Satellitosis was marked and glia elements were frequently seen within the cytoplasm of ganglion cells. Some of these parasitical-like glia cells showed regressive changes, but in general progressive changes were prominent. No lipoid substance was visible in some of the neuroglia elements in the vicinity of degenerated ganglion cells.

In certain parts circumscribed areas were intensely diseased, whereas the immediately surrounding region appeared almost normal. Such areas of degeneration could be observed to a greater or less extent around some of the vessels, which themselves appeared but slightly affected.

The adventitial spaces of most vessels traversing degenerated areas were enlarged and to a greater or less extent filled with disintegration products. This substance occurred in the form of yellowish pigment, staining with different degrees of intensity with basic aniline dyes. Lying within these masses a nucleus was frequently visible, which usually presented a pycnotic condition; a progressive vacuolar degeneration; or, in other instances, a polyhedral or triangular shape, characterizing it as the nucleus of a Körnchen cell. Ofttimes, in consequence of the compactness and intense staining of the granules, no nucleus was discernible in such accumulations. With the Herxheimer method the greater quantity of this material could be distinguishable as lipoid substances within the cytoplasm of Körnchen cells. However, large aggregations of a fatty character were discernible lying free within the lymph spaces. It was interesting to observe that such lumps usually presented a regular, roundish contour, but when closely packed together were exceedingly irregular in form, which



gave the impression that this substance may have been in a fluid or semi-fluid state before fixation.

In sections stained with toluidin blue some Körnchen cells were seen to contain basophilic metachromatic substances mingled with greenish tinged granules. Similar material was also perceived in neighboring glia cells. Many vessels and capillaries showed a considerable widening of the adventitial lymph spaces, which contained neither products of disintegration nor infiltrating elements.

The adventitial coat of some of the arteries was slightly thickened, but no trace of endarteritis could be perceived in any of them, and not a single obliterated vessel was encountered. Although these pathological changes were perceptible in the different nuclei of the thalamus, the nerve cells of the lateral nucleus appeared more intensely affected.

Examination of the lenticulate nucleus showed it to be similarly diseased.

With the Nissl method it was apparent that an unusually high grade degeneration of the ganglion cells was present in this region. Hardly a single cell could be found which did not exhibit marked pathological changes. In many of the diseased cells the changes in the nuclei were striking; vacuoles were discernible in the cell body, and the chromophilic substance was irregularly disposed in lumps or fine granules within the cytoplasm and processes. Numerous cells appeared as mere shadows surrounded by faintly colored granules. A pronounced increase in the cell pigment was a prominent feature of the degenerative changes in some instances; other cells were shrunken and sclerotic. The neuroglia tissue was greatly increased.

The cytoplasm of the glia cells lying between the nerve elements had undergone an almost incredible hyperplasia, with nuclear proliferation of a progressive character.

Regressive changes of the glia were also seen, frequently appearing as small nuclei lying within an enlarged cell body. A pronounced gliosis occurred in the vicinity of many capillaries.

Circumscribed foci of degeneration analogous to those found in the thalamus were encountered. In the degenerated areas many Körnchen cells were in evidence, their cytoplasm in most cases filled with yellowish, or yellowish-green, colored granules. Gliogenous phagocytic cells not infrequently occurred in groups, espe-

cially around capillaries, passing through or in the neighborhood of degenerated areas. Where many of these cells were collected about a blood vessel the enlarged perivascular spaces were seen to contain small and large irregular shaped masses of a yellowish tinge, and much peculiar material representing in part at least coagulation products of pathological tissue fluids. Apparently the cells extruded substances into the same space.

In these areas lipid granules were visible in the cytoplasm of the cellular elements of the vessel walls, especially of the adventitial lymph space.

No trace of endarteritis could be perceived in any of the vessels. Neither obliteration of capillaries, hemorrhages, proliferation of the vascular endothelium nor any evidence of sprouting new capillaries could be found.

A careful investigation of the fiber content of the basal ganglia, internal capsule and subthalamic region was made by the Weigert and Weigert-Pal methods.

In sections through the upper part of the right corpus striatum, the fine fibers passing mesially from the putamen were unaffected. The fibers of the internal capsule also appeared unaltered.

At a somewhat lower level, however, the internuncial fibers were diminished, and the medullary laminæ were defective, and atrophy of the nucleus was quite apparent. The external capsule presented a normal appearance. In the lower part of the nucleus the atrophy was more pronounced, the external and internal laminæ were considerably degenerated, the internuncial fibers were atrophic and some of them had disappeared. The internal capsule stained normally in all the sections and no degenerated fibers could be detected.

The slender tracts of fibers passing through the capsule and uniting the caudate and lenticulate nuclei were somewhat reduced in number, but most of them appeared normal.

Relative to the fibers of the optic thalamus a considerable diminution could be observed in the vicinity of the lateral nucleus, and a reduction in the fibers of the external medullary lamina was likewise visible. A number of the striothalamic fibers were degenerated.

In the subthalamic region it was evident that some of the nerve fibers of the zona incerta had undergone degeneration. Many of

the fibers of the lenticular loop in its course around the medio-ventral border of the basis pedunculi were degenerated. A number of the fibers of that portion of this tract which passes to the corpus Luysii were also affected. A deficiency in the fibers of the fasciculus retroflexus was apparent. The subthalamic nucleus was somewhat atrophic, but the majority of its fibers were fairly well preserved. The field of Forel appeared somewhat altered.

*Crura Cerebri.*—No defects in the crusta were visible in sections stained by the Weigert-Pal method. The fibers of the pyramidal tract stained deeply, and were not diminished in quantity. The mesial and lateral sectors were unaffected.

The tegmentum was apparently normal, but some of the cells of the substantia nigra were slightly altered. The fiber content of the nucleus ruber was impaired. A considerable number of the nerve cells were affected. In many of them various stages of chromatolysis was visible; others showed pronounced pigmentary degeneration.

Some of the cells were shrunken and their apical processes more tortuous than usual. Marked increase in the neuroglia elements was obvious, and proliferative changes in evidence. A gliosis occurred around many of the nerve cells which were intensely degenerated; and not infrequently only a few remnants of the cytoplasm were still discernible.

*Medulla Oblongata and Pons.*—No degeneration in the medullated fibers could be discovered by the Weigert method. The corticospinal tracts stained normally and no atrophy or loss of fibers was apparent. The fillet, olives, and restiform bodies also appeared normal. No characteristic degenerations could be found by the Marchi method. The cells of the motor cranial nuclei, with the exception of a few showed no appreciable deviation from the normal. Some of them were slightly shrunken, stained darkly and their processes appeared unusually wavy. In others a granular degeneration of the neurofibrils was perceptible. Similar changes occurred in a few of the cells of the nucleus cuneatus and nucleus gracilis. There were no changes in the pons that could be regarded as pathological.

*Cerebellum.*—A few of the Purkinje cells show chromatolytic changes, and neurofibrillar disintegration was visible in some cells.

The granular layer presented no departure from the normal, but a slight increase of glia cells was apparent in the molecular layer. No fiber decrease could be detected with the Bielschowsky or Weigert-Pal methods. A few of the cells in the corpus dentatum were shrunken and atrophic; but in general no changes were detected in the large triangular and stellate cells of this nucleus which could be regarded as pathological. No appreciable defect was discoverable in its rich plexus of nerve fibers.

*Spinal Cord.*—Slight diffuse connective tissue-thickening of the pia was present, but cellular infiltration was entirely absent, and no evidence of any inflammatory process could be discovered. The spinal vessels appeared normal; no thickening of their walls or endarteritic changes were discernible.

The cells were carefully examined in the cervical, dorsal and lumbar regions of the cord.

In the cervical region the motor cells of the anterior horns were well preserved. The majority were of good shape, the chromophilic bodies well defined and the nuclei centrally located. In these cells no abnormal increase in pigment or other pathological changes were visible. Some of the cells, however, were shrunken, stained more deeply than usual, and the typical arrangement of the Nissl elements disturbed. In the posterior horns it was also evident that some of the cells had undergone certain pathological changes, consisting in a slight shrinkage of the hyaloplasm, causing a more compact arrangement of the tigroid bodies, and in consequence a deeper-stained appearance to the cell. Such cells often had an elongated aspect and tortuous processes. The nucleus stained darkly, but otherwise appeared unaltered. A few of the cells of the lateral horns were slightly swollen and commencing chromatolysis was visible round the nuclei. The motor cells of the anterior horns in the dorsal region showed no distinctive alteration. In some of the cells of the posterior horns, however, a granular disintegration of the tigroid bodies occurred; others showed some pigmentary degeneration. In the lumbar region the cells of the anterior horns were well preserved. Here and there a cell was encountered in which the characteristic arrangement of the Nissl substance was affected, but otherwise no alteration could be detected. Pigmentary degeneration was discernible in a few of

the cells of the posterior horns; in others commencing chromatolysis was perceptible.

Longitudinal and cross sections were made from different levels of the cord for the investigation of the fibers.

The sections stained by Weigert's method were negative, and there was no indication of degeneration or shrinkage in any of the columns of the cord.

The direct and crossed pyramidal tracts showed no change. The medullated fibers of the gray matter appeared normal. With Marchi's method the picture was likewise negative. No evidence of systematized fiber degeneration was apparent in the cortico-spinal tracts in any part of the cord. A few typically degenerated fibers were encountered in longitudinal sections by the Alzheimer-Mann method.

*Peripheral Nerves.*—Sections from the ulnaris, radialis, medianus and ischiadicus were carefully examined, but failed to disclose any pathological alterations.

#### RÉSUMÉ OF THE ANATOMICAL FINDINGS.

The brain was firm, large and well developed. There was no evidence of atrophy, and the convolutions presented a normal disposition. The meninges were normal, other than an inconsiderable thickening of the pia-arachnoid in some places. The cerebral vessels showed no deviation from the normal. The lateral ventricles were not dilated and no ependymitis was present.

Microscopically, the pathological changes in the cortex of the brain were distinguished by alterations in the nerve cells and neuroglia tissue. These changes were approximately uniform in the cortex of the different lobes, but apparently most pronounced in the frontal region. The alterations in the ganglion cells were confined chiefly to the small and medium-sized pyramids, and were characterized principally by shrinkage, although various types of cell disease were in evidence.

In the motor region the giant cells of Betz were exceptionally well preserved.

The neuroglia tissue showed both progressive and regressive changes. A considerable increase in the number of small astrocytic cells was present. Deficiency of the tangential fibers was discernible in some areas. The white matter of the cerebral hemi-



spheres was normal; and the pyramidal system, traced from its origin in the Betz cells of the motor area throughout its course, presented no alterations of any significance.

In the optic thalamus intense degeneration of the nerve cells, with an enormous increase of the neuroglia tissue, was found in the external nucleus, but small circumscribed areas of degeneration were also discernible scattered through the ganglia. The lenticulate nucleus was similarly affected, but the degeneration appeared more diffuse. Many of the nerve cells had vanished, and a marked neuroglial hyperplasia was present. The changes in the caudate nucleus were less severe.

The vessels of the diseased areas showed an enlargement of their adventitial spaces, and in some instances slight thickening, but were otherwise unaltered.

The internal capsule was intact, but the striothalamic fibers were reduced. Some of the fibers of the lenticular loop were degenerated, and the subthalamic nucleus appeared somewhat atrophic. The fasciculus retroflexus Meynert was slightly affected. A number of the cells of the tegmental nucleus were considerably altered, and increase of the glia cells was perceptible.

No systematized fiber degeneration could be found in the spinal cord, and the motor cells of the anterior horns, with the exception of a few, presented a normal appearance.

The interesting features in this case are the early onset of the disease, the absence of homogeneous heredity, and the pathological changes.

Huntington, who was the first to give a full description of this disease, referred to its independence and differentiation from other types of chorea, especially Sydenham's chorea.

He laid especial emphasis upon heredity as a salient etiological factor, and the development of the disease late in adult life. While in general the disease does not usually occur until the third or fourth decade, it is obvious, from a review of the literature, that the affection may become apparent at a much earlier period in life. Menzies and others have observed that the age at which it appears frequently precesses generation by generation, but the exceptions are numerous. Heilbronner has suggested that the earlier manifestations of the affliction may correspond to a severer course of the disease.

An instance in which the disease began unusually early in life has been described by Friedenthal. Hoffmann demonstrated a case in which the first symptoms of the disease were exhibited in the twentieth year of the patient's life. Diller mentions 10 cases which developed before the twenty-fifth year; and Kölpin reported a case beginning in the twenty-second year. In a case published by Goldstein, the choreic movements became evident between the twenty-second and twenty-third years of age. Wollenberg also reported a case in which the disease began at the age of twenty, terminating in dementia. A number of other cases occurring quite early in life have been published.

Chronic progressive chorea is probably one of the most familial of all diseases, and heredity has been regarded as a significant etiological factor.

In the case under consideration the absence of hereditary transmission and degenerative stigmata is striking.

Among some instances in which no heredity could be ascertained may be cited the case reported by Frank. The patient was affected with the disease in her forty-third year; the choreic movements began in her right arm and gradually involved the entire body. The mental symptoms were characterized by irritability and a progressive deterioration of the intellect. Schabad describes a case which developed in a woman forty years of age, who had suffered for a number of years with attacks of migraine. No hereditary factors could be determined. Continuous movement of the head and unrest of the musculature occurred; extensive involuntary movements of the upper extremities and constant extension and contractions of the lower extremities were present. Excitement increased the symptoms. The movements could be influenced to a slight degree by the will, and practically ceased during sleep. Speech was affected and mental reduction evident.

In one of two cases reported by Westphal the cause of the disease was attributed to injuries sustained by the patient in falling from a scaffold. Shortly after his discharge from the hospital choreic movements developed, which subsequently increased in intensity. Speech was affected, the reflexes exaggerated and an apathetic progressive dementia supervened. In one of three cases described by Frotscher no heredity could be determined. The patient was struck on the head by a heavy beam and subsequently

developed chronic progressive chorea. Skoczynski reports the case of a woman who developed chronic progressive chorea following a severe fright, caused by a fire in the patient's house. No hereditary factors could be ascertained. A. de Castro, Bonfigli, and others have also reported cases in which hereditary factors were wanting.

In considering the morbid anatomy of chronic progressive chorea it is apparent, upon reviewing the literature, that many of the pathological observations recorded afford but meager data toward an explanation of its semeiological characteristics.

The unsatisfactory results are in part due to the fact that early cases seldom die and advanced ones are to a greater or lesser degree complicated by pathological tissue changes quite sufficient to conceal the slight morbid impairment presumably responsible for the symptoms, thus causing a lack of uniformity and a variation in the descriptions of the pathological alterations.

It therefore appears evident that chronic pachy- and leptomeningitis, hydrocephalus, œdema, and vascular changes assumed by some authors (Oppenheim, Facklam, Weidenhammer, Wollenberg), as the pathological basis of the disease, can hardly be accepted as enacting more than a casual rôle in its morbid anatomy, as similar conditions are not uncommonly observed at autopsy in arteriosclerotic and senile types of dementia, with which advanced cases of chronic progressive chorea are frequently associated. Hence cases of this description are obviously unsuitable for pathological investigation. It is therefore probable that in consequence of such terminal complications some of the earlier authors (Weidenhammer-Binswanger), at a time when the histological processes in paresis and senile dementia were obscure, believed that an analogy existed between these diseases and chronic progressive chorea. There are, however, at present a number of cases on record in which the pathological alterations observed could in no way be confounded with those produced by a syphilitic process. Likewise in our case there was no pathological lesion of the vessels, or meninges, which resembled what is found in syphilitic diseases of these structures, and the Wassermann test obtained during life proved negative. In view of these facts, there is no reason why syphilis should be advanced as a possible basis for this disease.

A frequent microscopical change observed by writers on this subject is a hyperplasia of the glia in the cortex of the cerebrum. This increase of neuroglia has been described either as a uniform process throughout the cortex, or more pronounced in certain layers, and varying considerably in its degree of intensity. Some investigators have reported a general increase of glia cells (Greppin, Modena, et al.). Clarke found the neuroglia increased in the second and third layers of the cortex. Collins reports a hyperplasia of glia chiefly in the deeper layers; Schulz an increase in the number of neuroglia cells in the layer of large pyramids; Menzies a coarseness of neuroglia in the first layer. Raecke found the glial increase most marked in the motor region. Stier describes a proliferation of glia cells principally in the layer of small and medium-sized pyramids, which he regards as a primary process resulting from an inherited anomaly of the cortex. A number of writers have therefore concluded that a hyperplasia of the glia constitutes the characteristic alteration in Huntington's chorea.

Lannois and Paviot proposed an explanation for the involuntary movements occurring in this disease, by the hypothesis that an irritation of the otherwise intact giant cells is produced by the proliferating glia, thus inciting them to excessive function. Keraval and Raviart, Kattwinkel, and more recently Margulies, have advanced similar theories. This hypothesis, however, seems most improbable, as it is difficult to conceive how an irritative reaction of the glia could continue for years, and the motor cells retain their integrity.

It appears obvious that undue prominence has been given by some investigators to the increase of the glia, as a factor in the pathogenesis of this disease. A careful review of the more important contributions to the pathology of chronic progressive chorea, together with the observations here reported, indicate that the hyperplasia of glia occurring in this condition can in no sense be considered a primary process, as opined by Stier, but that the changes in the neuroglia are secondary or reparatory in character, consequent upon degeneration of the neuron elements.

Structural anomaly in the architectonic of the cortex has been suggested by Kölpin as a possible foundation for the development of the disease. But this offers no adequate explanation for the



usual late manifestation of the symptoms and the progressive character of the affliction.

A diffuse degeneration of the ganglion cells of the cortex is the most constant pathological alteration which has been described in this disease. In some instances the nerve cells in certain layers were more intensely affected. Menzies found the degeneration of the cells most pronounced in the deeper layers; Rusk, Stier, and others the greatest variation in the small and medium-sized pyramids. The failure of most investigators to find any alterations in the Betz cells which could be regarded as pathological is significant (Greppin, Stier, Rusk, Kölpin, Raecke, Pfeiffer). Other writers have observed a variable amount of disease in the cellular elements of the cortex and basal ganglia (Modena, Margulies, Alzheimer).

In this connection it is interesting to note that the severity in the alterations of the nerve cells and the extent of defectiveness in the cortical fibers, seem to vary in proportion to the degree of dementia.

A slight marginal loss, or irregular degeneration, in a few fibers of the spinal cord has been observed, but such changes may occur normally, especially in aged individuals.

From the exhaustive anatomical study in the case here reported, we may conclude that the involuntary movements in this disease probably result from a primary degeneration of the neurons in the nucleus ruber, the lateral nucleus of the optic thalamus and the lenticulate nucleus, thus interfering with the conductive integrity of the cerebello-thalamo-rubro-cortical path.

As regards the brain and spinal cord, the motor cells were well preserved, and the cellular changes present were of a character similar to those encountered in conditions unassociated with choreiform movements. The pyramidal system was intact and no systematized fiber degeneration could be detected in the spinal cord.

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ON THE TOPOGRAPHICAL DISTRIBUTION OF CORTEX LESIONS AND ANOMALIES IN DEMENTIA PRÆCOX, WITH SOME ACCOUNT OF THEIR FUNCTIONAL SIGNIFICANCE.

(CONCLUDED.)

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IV. CLINICAL AND ANATOMICAL ANALYSIS OF TWENTY-FIVE CASES OF DEMENTIA PRÆCOX, BEING A RANDOM SELECTION.

At this point I shall present (a) the condensed *clinical history*, (b) a summary of the *autopsy findings* in the trunk and limbs, (c) a transcription of the *brain findings* both on the autopsy table and by means of subsequent review in the light of systematic photography, and (d) a provisional classification as to *congenital and acquired features* in the light of all available facts, in a series of 25 cases. This series contains three cases previously studied less systematically, and presented in 1910 as Cases XII, XIII, and XIV. Another case (1297) was mentioned in 1910, but was excluded on account of a cyst of softening (the study in 1910 deliberately excluded many cases on account of complicating features); but, as the brain of 1297 was available for systematic photographic study, no reason could suffice for its exclusion from the present series, and it is included as Case I.

The cases are presented in the chronological order of their appearance in the post mortem room, as this order seemed less likely to prejudice interpretation than any other.

CASE I.—F. L. (D. S. H. 6556, Path. 1297), female, of Nova Scotian stock, was regarded on admission at 29 years as a case of "*acute melancholia*," and in fact had had a previous attack of *depression* (with

hallucinations of hearing) at 20, which lasted 18 months and terminated in complete recovery. Marriage: three children, last at 27 years. Patient went home after six months and returned again a year later in a condition termed *chronic delusional insanity* (1894). There were considerable variations in the picture in the subsequent hospital course until death at 56 years. It does not appear that the diagnosis *manic-depressive insanity* was ever considered, despite the cyclothymic tendencies of the course.

The picture was one of so-called *dementia* varying in apparent depth from year to year, and difficult to evaluate by reason of the *deafness*, which, marked on admission, grew worse. It seems pretty well established that patient had *auditory hallucinations* of a reviling nature, and she gave evidence of reaction to these for many years by excited *outbursts of profane and at times obscene speech*. But patient *always denied hallucinosis*, except at first, when she admitted *voices* but said they *did not seem real*. At first, too, she complained of a *beating and a roaring noise in her head*. *Delusions* were *directed both at husband and at others*, especially *mother-in-law*. Not infrequently quarreled with other patients. A good worker in the sewing-room. Wrote numerous incoherent letters.

There was "*nervousness*" in her mother, who was also said to have been *given to talking to herself*. A half-brother was said to be insane. An aunt insane.

Physically, there was early a tendency to gain weight, despite poor appetite; but later patient remained thin and grew thinner, finally dying in emaciation doubtless increased by cancer of the uterus. The tongue was always *tremulous*. *Nasal catarrh*. Chronic changes in both drums were found at autopsy.

It seems clear that this case could be described symptomatically as *paranoic* and as *allopsychic* in Wernicke's nomenclature. There seem to be no evidences of *catatonia*, or perhaps *any true dementia*. Where can the case be placed in Kraepelin's classification (1904), save in the *paranoid group of dementia præcox*?

*Summary of autopsy findings*.—Cause of death: *Carcinoma of cervix and body of uterus, with infiltration of retroperitoneal tissues*. Aortic, aortic valvular sclerosis; mitral incompetency; edema of feet and of lungs; chronic passive congestion of liver; hydropericardium; enlarged mesenteric lymph nodes; large white kidneys; apical pleuritis; ovarian and mammary atrophy; ear-drums opaque.

The findings in the head were: Hair short and scant; brown, mixed with gray. Scalp very pliable, taking but little force to remove it. Calvarium of normal thickness and contains no diploe. Dura not adherent to skull-cap even by longitudinal fissure. Dura thin, the convolutions easily discernible through it. Light adhesions between it and the pia. The pia is hazy along vessels. The hemispheres are equal in size; but the sulci in the motor regions, on the left side particularly, gape to the extent of 1 cm., in frontals 0.5 cm., and gaping occurs on the left orbital surface of the frontal lobe. There is an area of softening on the right temporal second convolution, 2 x 3 cm. in diameter. The right temporal lobe is

sticky to the touch. The convolutions are very superficial and thin. A recent thin white fibrinous exudate encircles the pons. The cerebellum is very much softened; weight 180 gm. Basal vessels not notable. Brain weight 1390 gm. Brain hardened *in toto* in normal formalin. Middle ears opaque. Lateral sinuses contain much blood. Cord normal. Summarized: Calvarium dense and dura slightly adherent; slight sulcal chronic leptomeningitis; left-sided cerebral atrophy; recent focal lesion of right second temporal convolution.

The *unequal atrophy* of the two cerebral cortices was most striking on the autopsy table, and the systematic review by photographs only confirms the impression. Although perhaps no area is quite free from gross atrophy when viewed from some aspect, it would appear that the entire *left supra-Sylvian* region, involving more especially the *frontal, central, and parietal areas*, is most extensively affected by the atrophy, although (if one views the right hemisphere from above) the posterior part of the *right first frontal sulcus* gapes widely and deeply, suggesting that the maximal atrophy may lie here. A study of the two mesial aspects, however, again leaves us with the impression that the left hemisphere is a little more atrophic than the right.

The superior aspect intimates that the *post-Rolandic* tissues on the *right* side are *more atrophic* than on the left. The coronal sections confirm this intimation, more particularly for the convolutions near the parieto-occipital fissure.

It is noteworthy that, despite the well-marked cortical atrophies, there was little sign of diminution in the section-area of the corpus callosum. In particular there was very slight presplenial thinning.

Also, despite the long-standing and slowly increasing deafness, the temporal convolutions failed to show convincing lesions, not, at any rate, such as to surpass those of many other areas. Nor, I assume, can the cyst of the right second temporal convolution be invoked to account for the deafness.

A woman, depressed and hallucinated at 20, depressed at 29, deluded and variously "demented" from 30 to death at 56. Outbursts of profane and obscene speech and of quarrelsomeness. Delusions allopsychic (against family). Tremor of tongue. Noises in head, deafness (peripheral).

The brain appearances seem to place this long-standing case of paranoid dementia *præcox* pretty definitely outside any possible temporal-lobe group of paranoic conditions. In any case, the hallucinosis was early and fleeting, and may perhaps have been related with the early peripheral deafness. As to paranoic correlations, we find supra-Sylvian atrophy, more marked on the left side. The allopsychic delusion-formation could then be correlated with either frontal or parietal disorder. It would perhaps

be pressing correlations overmuch, should we try to correlate the outbursts of (catatonic?) excitement with something in the parietal lobe, leaving the paranoia as frontal. The profane and obscene speech and the incoherent letters are consistent with the left-sided atrophy.

However, the chief value of the case lodges in the demonstration of a *non-temporal* example of *paranoia*, paranoia in which doubtless hallucinosis was not the essential agent but which consisted in a false attitude to society. On general grounds (though not on the data of this case) one might suppose this false attitude rather a frontal-lobe affair than due to disorder more posteriorly.

CASE II.—From my previous study (1910) the following history is reproduced:

"XII. Woman, mill-worker (father alcoholic), at 23 had insomnia, followed by auditory hallucinations, fear, religiosity, vagrancy. In hospital, resistive, hallucinated, delusive, mute, refusing to eat; later, violence. Finally apathy, with manneristic speech and grimaces. Death from carcinomatosis, 21 years after onset. Occipital microgyria. Chronic leptomeningitis along sulci. Cervical spinal cord firm. (*Acquired.*)"

A revision of the brain findings in the light of the present systematic study is as follows:

The *left superior frontal region* presents what looks at first like an annectant, lying in a hollow about 0.5 cm. deep from the general contour of the lobe, but with normally thin pia mater investing it. This apparent anomaly is the more striking by reason of the massive gyrus co-ordinate therewith on the right side. Both grey and white matter appear unduly narrow in this small gyrus. Tracings of the two superior frontal regions show that the surface included between co-ordinate sulci is greater on the right than on the left side.

The frontal and temporal opercula on the left side flare more than on the right, and the posterior portion of the left superior temporal gyrus disappears in the bottom of the Sylvian fissure at the anterior end of the posterior third. Frontal sections through the brain suggest that both temporal and both hippocampal regions are less well supplied with white matter than the remainder of the brain. But the left temporal region appears least well off in this respect, and the pia mater, though not appreciably thickened, peeled with some difficulty from the region, suggesting, though nowhere definitely showing, adhesions. Comparison of the two superior temporal gyri is specially tempting.

A woman, sleepless, hallucinated, apprehensive, religious at 23; later deluded, resistive, mute, sitophobic, violent; finally apathetic, manneristic, grimacing. Course 21 years.



Unlike Case I, Case II cannot exclude the temporal lobe from a share in the production of paranoia. The brunt of the lesion was undoubtedly borne by the left superior frontal and left superior temporal convolutions, both of which suggest congenital or early maldevelopment. The maldevelopment of the frontal region was probably of the nature of an interference with the development of tissues properly laid down embryologically, suggesting *hypoplasia* rather than the *paraplasia* which the temporal lobe suggests. Auditory hallucinations can perhaps be correlated safely with some sort of (functional or structural) disorder of the temporal regions.

We might accordingly correlate the hallucinosis with temporal-lobe disorder, the paranoia with frontal-lobe disorder, and the late catatoniform symptoms with a later involvement (microscopic?) of post-Rolandic tissues (occipital-lobe anomaly is suggested by the gross appearances). The case is obviously too complex for the exact correlation of symptoms with lesions; but there is in the case nothing inconsistent with the general contentions of the work in 1910 or with those of the present work.

CASE III.—From my previous study (1910) the following history is reproduced:

"XIII. High-grade imbecile. Irish mill-operative. Sister epileptic. Mother of three children and a fourth illegitimate. One attempt at suicide? Eight years' course, with onset at 23. Religiosity. Could see Virgin Mary at any time (hallucination rather than delusion). Imitation of saints' attitudes as seen in pictures. Possible gustatory hallucinations. Death at 31. Unequal pupils. Superior temporal anomaly. Cervical spinal cord unusually large. Slight leptomeningitis, especially basal. (*Acquired and congenital.*)"

A revision of the brain findings in the light of the present systematic study is as follows:

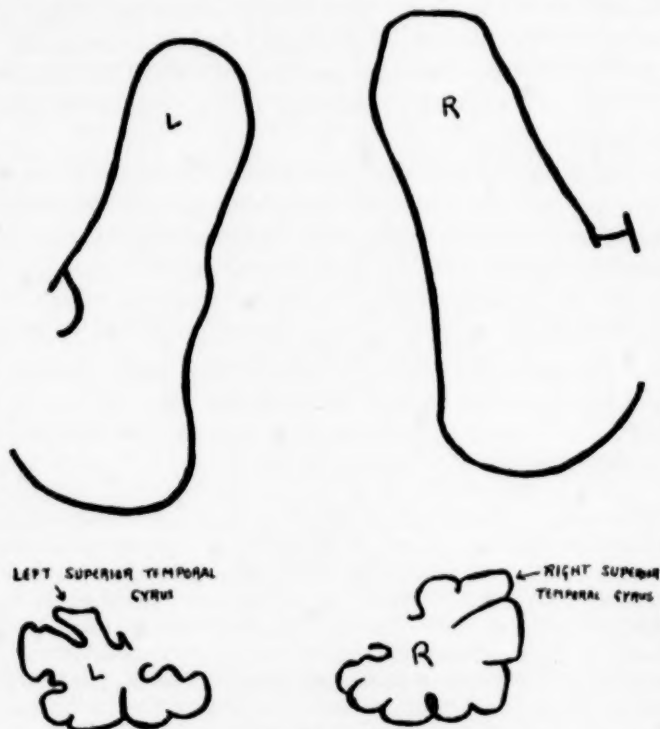
The most striking feature of this brain—and that which caught attention in the former publication, where this case appeared as Case XIII—is an anomaly of the left superior temporal gyrus, which is almost entirely hidden in the middle part of its course by the middle temporal gyrus. Tracings of the contour of the two temporal lobes, laterally viewed, suggest that there is an actual undersize to the left region as compared with the right. (Text-fig. 1, p. 608.)

Tracings made from appropriate frontal sections demonstrate the same fact. (Text-fig. 1, p. 608.)

It is clear that the amount of white matter in the temporal lobes is far less on the left side than on the right side.

The lateral ventricles are moderately dilated, but the left much more so.

A woman, regarded as possibly a high-grade imbecile, showed hyperreligiosity at 23. Voluntarily induced visions (Virgin Mary). Assumption of saints' attitudes. Religious songs sung.



TEXT-FIG. 1.

Here the correlations are not too strongly in evidence. Clinically we must remain in doubt whether the attitudinizing was catatonic or paranoid in origin. In fact, the history is not full. I get the impression, however, that there were numerous sensorial elements in the case (bad tastes, visual hallucinations). There was considerable amnesia (imbecility, however). The moderate *internal hydrocephalus*, more marked on *left* side, coupled with the *left-temporal-lobe hypoplasia*, suggests more a correlation with

certain imbecilic features than a correlation with the acquired mental disorder. We should greatly desire to find in the history of this case some auditory basis for her symptoms. On the whole, however, there is more visual basis than auditory.

CASE IV.—From my previous study (1910) the following history is reproduced:

"XIV. Shoe-operative. Attack of doubtful character at 12 years. Paranoid symptoms began at 18. Catatonic symptoms gradually supplanted the paranoid. Death at 32. Spinal cord unusually small. Brain weighed 985 gm., but was apparently not to any great degree atrophied. Heart (145 gm.) and liver (945 gm.) also small. (*Congenital.*)"

A revision of the brain findings in the light of the present systematic study is as follows:

The brain (weight 985 gm.) of this case was first described—under Case XIV of the former communication—as "not to any great degree atrophied." On account of the microcardia and microhepar, the suspicion was that the case was congenitally defective (this despite the fact that the subject is said to have been an adequate shoe-operative). More extended study calls attention to the fact that the occipital, and perhaps still more the temporal, regions are more atrophic (or aplastic?) than the rest of the brain. This difference is noticeable both in the external appearances and in the frontal sections.

A man, confused (and unconscious?) at 12; sleepless, "mesmerized," violent, suicidal, at 18; fantastic delusions, fantastic transitivity; profanity, obscene speech; later pronouncedly catatonic (special attitude).

Correlations cannot be made on account of universality of lesions. If anything, the paranoia might be regarded as post-Rolandic or sensorial in origin. Possibly the fantastic type of delusions will prove more often post-Rolandic than pre-Rolandic; for such delusions are on general grounds more nearly allied to normal exercise or overexercise of the imagination than to the assumption or establishment of an abnormal attitude to normal ideas. Thus, on general grounds we may conceive a more "impressional" type of delusions and a more "expressional" type, the former more apt to be post-Rolandic, the latter more often pre-Rolandic in origin. The patient under consideration obviously had the power of conceiving and probably of imagining more complex ideas and situations than a normal person. Suppose the post-Rolandic and infra-Sylvian emphasis of the lesions in this case to be roughly correlative with the fantastic (sensorial) delu-

sion-formation; then the later development of catatonia would be not unnatural, because kinæsthetic disorder would be apt to ensue from a spread of the process to near-by parietal regions.

The case is surely allied to cases of the new group *paraphrenia phantastica*, which Kraepelin has separated out from the other cases of dementia præcox. Kraepelin, however, remarks that about 40 per cent of his cases of dementia paranoides (as described in the seventh edition, 1904) turn out essentially cases of dementia præcox in the ordinary sense, and accordingly do *not* turn into the new (1913) group of paraphrenias (analysis of Zerko). Perhaps, then, we would best regard this case as one of paranoid dementia præcox, suggesting *paraphrenia phantastica*, but proving by its outcome its closer relation to dementia præcox (*i. e.*, by the catatonic features of the terminal condition).

See further discussion under Case VII.

CASE V.—M. W. (D. S. H. 13704, Path. 1383) was an old Scotch woman, a pensioner (husband in Civil War), of whom all history was lacking, but whose mannerisms, ways of thinking, and amnesia suggested the diagnosis *dementia præcox*. The examiners were inclined to suspect an *alcoholic* admixture in the dementia. Her age even was doubtful, but was at least 62 at death.

Untidiness, resistivism, loquacity, repetitive talk, loud singing, perseverant "Open door, open door," spasmodic movements of lower jaw, lid-closure, violence to patients, mischievous acts, silly smiling, gluttonous eating, disorientation for time and place.

Occasional more lucid intervals with euphoria and long narrative conversation about mantel-piece, tea-canister and a clock once possessed by patient.

Death at about 62 from *bronchopneumonia*.

*Summary of autopsy findings.*—Emaciation; general arteriosclerosis; various chronic lesions: cystitis, nephritis, apical tuberculosis, pleuritis, pericholecystitis, perisplenitis. Chronic internal hemorrhagic pachymeningitis of right side.

Head: Dura markedly adherent to calvarium on right side. Brain weight 1060 gm. (60 gm. loss, according to Tigges' formula). Marked diffuse chronic leptomeningitis, most marked over right Sylvian and Rolandic regions. The moderate atrophic process, like the thickening of pia mater, is more marked in pre-Rolandic tissues and on the right side. The frontal atrophy is better marked mesially than laterally. The sulci at the junction of the right superior and middle frontal gyri are deep. Both Sylvian fissures and the right first temporal sulcus are deep. The corpus callosum is thinned in an unusual place (in the present series of examinations), viz., in the region just back of the rostrum. Moderate to marked internal hydrocephalus, especially of mid portions of ventricles.

A woman whose psychosis is classed as dementia præcox on the basis of the terminal condition. The diagnosis is accordingly dependent on accepting at its face value Kraepelin's statements about characteristic end-states. No suspicion as to the duration of this case can be risked. There was such a general distribution of lesions in the areas with which we are particularly concerned—frontal, postcentral (Rolandic) and temporal regions—that no correlations can be safely made. There is, however, in the findings, nothing inconsistent with our general contentions. Such long-standing cases are rarely suitable for correlations.

We have no definite evidence of hallucinosis or delusions in this case; but the case should not be counted one way or the other in topographical analysis. Its chief or only value must lie in showing the nature and spread of the mild lesions that may be shown by a terminal case of what was probably dementia præcox. The suspicion of alcoholism must be remembered in connection with the general Rolandic (precentral as well as postcentral) atrophy.

CASE VI.—C. B. (D. S. H. 14779, Path. 1413) was a boy of 15 when he was committed to D. S. H., and died there a year and four months later of typhoid fever, ulceration, and peritonitis. The mental diagnosis was eventually considered to be dementia præcox.

Mother nervous and melancholy during her pregnancy with this boy; hard usage and neglect by husband complicated the pregnancy. From an accident to the boy at seven, is said to date a change of character, with refusal to go to school, fretfulness, and nervousness. At 14 another accident to back and head. An inmate of a truant school for two periods of three and two years respectively. Uncle a patient at state farm.

Auditory hallucinations, refusal to eat, nervousness and certain delusions antedated commitment by only a week. (Masturbation had begun four or five months before, but had probably ceased.) Insomnia is said to have lasted four nights after onset. *Food poisoned. Passers-by or Indians were to kill him* (given to blood-and-thunder stories). "*Had never done anything to gladden America,*" "*Traitor,*" "*Going to be shot.*"

Cyanosis of hands. Gait staggering (no Romberg); right cremasteric reflex not active.

After eight months' observation it was thought that the patient really belonged in the moron or subnormal group of defectives, and in the quasi-criminal class. A little over 11 months after the delusional episode upon which he was committed, patient developed a spell of disturbance which lasted 10 days. In this spell, patient was noisy, threatening, profane, denudative, given to disconnected and incoherent talking, lost weight and grew pale. Almost three weeks later typhoid fever developed, and death followed from peritonitis.



*Summary of autopsy findings.*—Aside from the typhoid fever lesions, there were bronzing of skin; unusually small adrenals (questionable glycosuria had appeared on entrance, but later disappeared); a small thyroid; slight sclerosis of aorta and right auricular endocarditis; cardiac hypertrophy; chronic myocarditis, pleuritis and pericarditis.

The brain weighed 1435 gm. (over weight 90 gm. by Tigges' formula). There is nevertheless a tendency to *frontal atrophy*, perhaps more marked on *left* side. Striking anomalous folding of convolutions in both callosomarginal regions above the splenium; but on the left side the fissuration suggests acquired lesion rather than congenital anomaly (region above and behind the rostrum).

A boy, truant school inmate five years; hallucinosis, refusal to eat, delusional episode at 15. Possibly moron, or subnormal, or quasi-criminal. Some symptoms of pluriglandular disorder. Catatonic excitement for 10 days.

On account of the comparatively brief duration of this case (16 months), it might well be imagined that the brain would not have registered coarsely any extensive lesion. The brain was, as noted above, probably somewhat over weight. There is a tendency to bilateral (more left-sided) frontal atrophy or aplasia, which may perhaps be correlated with the conduct-disorder, and with which in some partial way the delusional episode could perhaps be connected. More interesting is the callosomarginal lesion, which so far suggests an acquired feature.

This case is to be subject to especial analysis from the moron standpoint.

CASE VII.—F. C. (D. S. H. 8161, Path. 1472) was a case of so-called "primary delusional insanity" in a teacher with normal-school education who slowly developed *fears of abduction*, and already at 28 had *fear of her murder by Catholics*. At 30 was examined by alienist and described *hearing signals and seeing strange actions* (ideas of reference rather than hallucinations?). *Soul swept 10 feet from her body by stream of electricity. Tormented with electricity by persons under the eaves. Mother poisons the boarders. Children taken into a house to have heads compressed.*

After admission at 40, soon became somewhat euphoric and very busy. Delusions concerning special treatments which she is receiving (hypnotism, massage, moulding of head). If she got a *newspaper* from a certain patient, it was *really a million dollars for which she was selling her soul*. Stopped eating meat and said she had acquired "*the thought language, i. e., false hearing, a divine inspiration*," also had become able to talk with *beasts, birds and insects*. Parents had brought up children for scientific experiments and given them away.

Aside from refusal of food on the basis of delusions, no actions surely interpretable as catatonic developed until 48 (peculiar stepping movements).

At 50, frequently hallucinated (*Jesus standing by*). Formerly deluded concerning marriage to a Mr. King, now she was married to the Prince of Wales, later to Christ. Carried bundles tied to waist, as she had no little children pulling thereat.

Purulent sinuses of breast at 54. Body weight reduced to 63 pounds.

*Summary of autopsy findings.*—The cause of death was *phthisis pulmonalis, with necrosis of ribs and cold abscess of breast; tuberculous enteritis, with ulceration; tuberculosis of spleen; mesenteric and general enlargement of lymph nodes; sacral decubitus; acute nephritis.*

Among chronic lesions were atrophic breasts, uterus, and ovaries; enteroptosis; pericholecystitis; perisplenitis; pleuritis; myocarditis; renal infarct.

The brain weighed 955 gm., a reduction of 220 gm. if Tigges' formula can be applied. The brain was described as "infantile" by the pathologist, Dr. A. H. Peabody, although from the clinical history it seems clear that the early education of the subject required more than an infantile brain. As against aplasia, the clinical history certainly suggests that we should be dealing with atrophy. Perhaps the *increase of consistence* noted on the autopsy table is in line with the diagnosis atrophy. There is little or no dilatation of ventricles, and the corpus callosum appears in good proportion to the remainder of the brain. The hemispheres are somewhat peculiarly shaped in that the occipital poles are much tapered off. The gyri bordering the Sylvian fissures are more atrophic than the rest of the gyri; next, perhaps, the middle frontals.

A woman with a 26-year course of paranoic symptoms beginning at 28. Hallucinosis or possibly ideas of reference of auditory content. Movements possibly catatonic at 48.

Whether this be a case allied to *paraphrenia phantastica* or a case better termed dementia paranoides (in the sense of Kraepelin, 1913), we must be interested in the fact that the case agrees in several respects with Case IV. Both cases show remarkably atrophic or aplastic brains (IV 985 gm., VII 955 gm.). Both cases have a history of complete social adequacy (IV a shoe-operative, VII a normal-school graduate teacher). Both agree in the possession of temporal-lobe atrophy (or aplasia?) more marked than that elsewhere. Both possessed delusions of a type which I provisionally termed (see Case IV, above) "impressionist" or sensorial, and presumably more closely related to post-Rolandic and infra-Sylvian cortex than to pre-Rolandic supra-Sylvian tissues. There was a tendency to greater atrophy of the middle frontals than elsewhere, in Case VII.

CASE VIII.—H. P. (D. S. H. 16166, Path. 1487) was a case regarded as a *catatonic dementia præcox*, although the whole course lasted about three weeks, terminated by *diphtheria*. There were no symptoms referable to the diphtheria, except some choking sensations a few hours before death at 34 years. (The diphtheria affected the tonsils and epiglottis, and the typical organism which was recovered was able to kill guinea-pigs in 24 hours.) The onset is said to have been with brooding over a death in the family.

The attack started with religiosity ("lost," "very bad") and talk about God and the devil. Excitement and occasional violence.

In hospital: resistivism, mutism, confusion, involuntary passing of water and feces. Looked worried, anxious, surprised. Could rarely be made to lie down. Insomnia, anorexia. At first the pulse was 120, high tension and volume (during last three days, pulse became normal). General tremor of the body, especially of the lips. Excessive growth of hair in axilla, hair over sternum. Obese.

*Summary of autopsy findings.*—Cause of death: *Diphtheria, bronchitis, acute pleuritis*. Also patent foramen ovale, persistent thymus, antelexion of uterus, asymmetry of face.

The brain (weight 1300 gm.) was asymmetrical (to correspond with the face), and showed especially a somewhat acutely tapering left occipital lobe (less marked, right). The frontal regions were full and fairly complex (exceeded by six other cases, right; but by 11 other cases, left). All the sulcus counts showed the right hemisphere more complex than the left (five areas). The fore-and-aft measurements showed the pre-Rolandic region far in excess of the post-Rolandic.

Despite these features, no focal emphasis of any lesion could be made out in the gross (neither sclerosis nor atrophy); and, while from an anthropological point of view doubtless far from negative, the brain must be regarded, from the narrower pathological point of view, as *negative*.

A woman of 34, with a total course (the shortest in the series) of three weeks, terminated by diphtheria. The case was regarded as *catatonic dementia præcox*, though perhaps the diagnosis is forced. A brief period of delusions was succeeded by symptoms of an exhaustion character among which resistivism and mutism probably gave rise to the diagnosis *catatonic*.

Whatever the correct diagnosis (it is even conceivable that the case is an obscure example of symptomatic psychosis), there seems to be some anthropological evidence (asymmetry) for supposing a congenital or early acquired basis for the development of *dementia præcox*.

CASE IX.—C. P. (D. S. H. 15546, Path. 1491) was a woman of wholly unknown antecedents, who died after a little over a year of symptoms hardly interpretable except as *catatonic*. Presumably a case of *dementia præcox*. Death at 53.

Persistent posture in bed, head held at right angles to trunk, eyes staring fixedly at space. Mutism, cerea flexibilitas, resistiveness, occasional negativism, eyelids not closing on approach of finger, untidiness. Before admission she admitted some ideas of being poisoned, but after admission was never got to say more than "I don't know. I can't tell you."

*Summary of autopsy findings.*—Cause of death: *Hypostatic pneumonia*. Bronchial lymphnoditis; bilateral obsolete tuberculosis and fibrous pleuritis; chronic pericholecystitis (old operation scar) and interstitial hepatitis; chronic gastritis and enteritis; mitral and aortic valvular endocarditis and chronic epicarditis; aortic sclerosis and sclerosis of coronary, internal mammary, external iliac, and basal cerebral vessels; umbilical hernia; retroversion of uterus; left ear-drum opaque.

Head findings: Dense thick calvarium except in fontanelle region. Dura and pia normal. Brain weight 1330 gm. (over weight by 50 gm. according to Tigges' formula). This brain was examined *three days post mortem*. The hippocampal gyri were firm; the occipital, frontal, and temporal regions were all softer (and in that order).

Systematic examination of the brain showed the left hemisphere longer and shallower than the right. The tip of the left temporal lobe is pointed. Section showed presplenial thinning of the corpus callosum. The mesial views suggest atrophy, particularly about the paracentral lobule. Chronic internal hydrocephalus.

A woman dead at 53, whose psychosis is classed as catatonic dementia præcox on the Kraepelinian basis of characteristic terminal state (compare Cases V, XV). An over-weight brain was coupled with a marked internal hydrocephalus.

Correlations need not be pushed, seeing that the history is obscure. In the history, however, we have some little evidence of delusions as well as marked catatonia. With the latter we may possibly correlate the mild atrophy (aplasia?) of the paracentral lobules.

The case will be further considered under the heading hydrocephalus.

CASE X.—M. P. (D. S. H. 15083, Path. 1507) was a case of somewhat doubtful diagnosis regarded as *paranoid dementia præcox*. There was an *hereditary taint* (paternal uncle a suicide at 36, an aunt insane for 30 years, a brother a suicide at 21). Menstruation at 20. Onset of mental disease resulted from a love affair. Inmate of two hospitals in New York State, from 24 to 28. Afterward unimproved, melancholic. Admitted to D. S. H. at 58, apathetic, deluded concerning brother and his wife. Masturbation observed at times. Mannerisms of speech.

Death at 60 from *generalized carcinosis*, a year and two months after removal of breast and pectoralis. Metastasis in brain.



*Summary of autopsy findings.*—Emaciation; scar, thorax; rigor mortis; edema, legs; arcus senilis; adhesions, appendix and spleen, colon, liver, lung; arteriosclerosis internal mammary, bicuspid and mitral valves, aorta and internal iliacs; bronchopneumonia; carcinoma of lungs, liver, brain; interstitial nephritis.

Head findings: Calvarium thin and soft. Edema of brain. Brain weight 1280 gm. Pons and cerebellum weight 145 gm.

Small depressed areas in postcentral gyrus bordering on median longitudinal fissure. Inferior temporal convolution on each side somewhat softer than usual. In one of the blood-vessels in the postcentral sulcus, right side, is a small amount of granular material. Brain throughout very edematous. Sulci show a slight tendency to gaping, especially in the temporal region. Floor of fourth ventricle smooth. Middle ears negative. Cord shows no gross abnormalities except a marked constriction in mid-dorsal region, which may have been caused in removing.

The third right frontal gyrus, anterior to temporal tip, contains a tumor 1 cm. in diameter, in the grey matter, and easily enucleated. In the precentral gyrus (right), superior portion, are two—the smaller 0.5 cm. in diameter, extending to white matter; the other 1.5 cm. in diameter, on the surface and extending 2 cm. into the cortex. In the precentral (right), lowest portion, is another, 1.5 cm. in diameter, not visible on surface but extending into the grey matter. Hippocampal gyrus (left), 1 cm. in diameter, tip. Occipital lobe (left), 1 cm. in diameter, lobus cuneus.

A woman who began to menstruate at 20, and who appears to have been insane from 24 to death at 60. Masturbation observed after age of 58.

Correlations are difficult by reason of the intercurrent brain metastases. Aside from these, the temporal regions appear to be anomalous and unduly simple in architecture. Only one other case (1574) has as simple a construction. This is the more remarkable, because the other regions whose sulci were counted are well up to the normal in complexity. The observation at autopsy of gaping sulci in the temporal regions emphasizes the simplicity of convolutional pattern here mentioned. The suggestion is that the region was laid down in the embryo with undue simplicity and later became subject to a moderate differential atrophy. No evidence concerning hallucinosis in this case has been as yet obtained. The frontal regions suggest an atrophy or aplasia of milder degree.

CASE XI.—G. P. (D. S. H. 10378, Path. 1509) was a case of *catatonic dementia præcox*, with onset at 21 of "acute melancholia." Data available concerning years from 48 to 58. The "attack" at 48 was said to be the sixth.



Hyperreligiosity, exaltation or indifference, excitement and impulsive acts, mutism, resistivism, negativism, refusal of food, mannerisms, characterized the last 10 years. Once secreted knives from dining-room on his person. Auditory hallucinations were suggested by some of patient's attitudes. No cerea flexibilitas, but for days at a time limbs were absolutely limp, opposing neither active nor passive resistance to motion.

Autopsy 24 hours post mortem.

*Summary of autopsy findings.*—Cause of death: *Tuberculous enteritis and miliary tuberculosis of lungs (also pleuritis) and kidneys.*

Acute or active lesions: Acute cystitis; sacral and left gluteal decubitus; abrasions and ecchymoses of right eye; edema of brain.

Chronic lesions: Emaciation; lymph-node enlargement, mesenteric, bronchial, inguinal, epitrochlear; peritoneal and pericardial effusion; aortic, coronary, internal mammary, pelvic arteriosclerosis; chronic pleuritis, pericarditis, perisplenitis, pachymeningitis; anæmic bone marrow of femur; fatty and cirrhotic liver; chronic mitral and tricuspid endocarditis.

Anomalies: Left middle finger amputated; asymmetry of face (left eye and ear elevated); patent foramen ovale; fetal lobulations of spleen; constriction of stomach.

Nervous system: The brain, by Tigges' formula, should weigh 1365-1370 gm., but actually weighs 1265 gm.

This brain shows a moderately well-developed corpus callosum with little or no trace of presplenial thinning. However, there is a distinct though moderate dilatation of the lateral ventricles. This dilatation probably corresponds with a moderate general atrophy of the frontal, central, and parietal, as well as the greater part of the mesial convolutions, about equal on the two sides. The anterior part of each superior temporal gyrus is somewhat withdrawn from the surface of the adjacent gyri.

A man dying at 58 after a course of 37 years. Correlations cannot be safely made on account of generalized lesions (see also below under hydrocephalus). Paranoia, catatonia, hallucinosis, were all clinically in evidence; ample and suitable lesions exist for correlation.

CASE XII.—M. C. (D. S. H. 8469, Path. 1518) was a woman stated to be hereditarily insane. "Dementia" showed itself at 16. Hospital treatment 20 to 22 (suicidal). Again under medical care (depression) at 26 and admitted to D. S. H. Emotional, capricious, abrupt in talk, once attempted suicide (masturbation). Boarded out two months until again restless and without appetite. On return, improvement. Periods of a few days of persistent masturbation. Periods of excitement. For some years would at times tear paper up in little bits and scatter it about. At 41 had become apathetic and careless about person. High-pitched, unmodulated, whining voice. Asked how she was, replied "I don't want all my teeth pulled." Asked how she was getting along: "It's fair weather." At 43 negativistic, resistive, laughing to herself, answers (except a very few) irrelevant,

destructive to clothing, scolding. At 44, acquired a habit of going to wash-room to wet hair, dress, and skin, refusing to go to bed. Phthisis had begun at 42. Death at 45.

*Summary of autopsy findings.*—Cause of death: *Ulcerative gastritis and enteritis.*

Acute lesions: Subpericardial hemorrhages.

Chronic Lesions: Emaciation; tuberculosis of left lung; chronic nephritis; aortic and internal mammary arteriosclerosis; mitral endocarditis; cholelithiasis; fatty liver; healed decubitus; fibromyoma of uterus.

Nervous system: Brain weight 1385 gm. (over weight by Tigges' formula,  $8 \times \text{body length, } 160 \text{ cm.} = 1280$ ). No lesions or anomalies were observed at the autopsy table, but on median section certain features were discovered. The most striking features of the brain are:

(1) A marked *presplenial thinning of the callosum* (which in general, however, is well-developed).

(2) An unusual appearance of *puckering and wrinkling of the right callosomarginal gyrus* in the long horizontal portion superior to the main part of the callosum (the latter condition resembles in a lesser degree the condition in 1413). The corresponding region of the *left callosomarginal gyrus* in 1518 shows several small sulci not found on the right; it is accordingly hard to avoid the hypothesis that the condition of the right depends on some developmental, congenital, or early factor. Its superficial resemblance to the gross appearances of certain other convolutions in certain cases supposed to have been victims of *early encephalitis* suggests a similar hypothesis for this case.

(3) The right hippocampal region both is superficially less extensive than the left and also, on cross-section, exhibits a retraction from its proper plane, due largely (it would appear) to the smaller size of the right cornu ammonis but perhaps also associated with a thinning out of the hippocampal white matter.

(4) There is a definite though slight degree of *chronic internal hydrocephalus*, more marked in the posterior cornua of the lateral ventricles. There is, however, no appreciable difference in the degree of dilatation on the two sides—though this might perhaps have been predicted if the causes of the hydrocephalus acted simultaneously with those of the hippocampal irregularities mentioned under (3).

(5) Very striking, in relation to the *right-sided hippocampal aplasia*, is a *left-sided frontal aplasia*. Counts of the sulci show that the *left frontal region* is *simpler* than the right (16:19), despite the fact that it is *smaller in volume* than the right. The other regions counted show no marked differences.

This case is to be subjected to more special study.

A woman with 29 years of symptoms, at first coming in attacks, later permanent. It is a question where to classify the case; on the whole, it would appear that the case became in the end catatonic. However, there were numerous evidences of paranoia.

I will not attempt to make correlations in this case, by reason of forthcoming more extensive studies. The peculiar schizophrenic irrelevance of replies should perhaps demand a temporal-lobe correlation not specially indicated in the above description; but the hippocampal disturbance may possibly involve the temporal region in ways which future study will reveal.

CASE XIII.—L. W. (D. S. H. 12692, Path. 1540) was a Newfoundland table-girl of Irish extraction, Catholic, who began to grow careless and to show poor judgment in little ways at about 20 years, but first showed pronounced mental symptoms some two months before admission at 23. Five years later epithelioma of the tongue developed and death followed in some nine months at the age of 29. It is not clear that this epithelioma had at all to do with the mental symptoms, despite the appearance of a small metastasis in the pituitary body (no glycosuria at any time, although no tests later).

The onset was with headaches, erratic conduct, apparent apathy, and delusions possibly of a sexual nature touching a mere acquaintance. Patient preserved an evasive and reticent attitude regarded as paranoidal until death, and remained as a rule quiet and apathetic, with occasional periods of irritability and restlessness. Careless as to dress, she was as a rule tidy in personal habits.

Impulsive striking at patients or attendants was attended with facial flush. Profane, not obscene.

Auditory hallucinations were surmised from certain reactions. Occasional talking to herself, silly, smiling, lying prone on couch, or standing for long periods against the wall. Was occasionally got to work on the ward or in the laundry for a few days.

It is interesting that, in addition to a fine tremor of extended tongue and fine tremor in handwriting, Dr. F. Robertson Sims found exceedingly lively knee-jerks and a bilateral ankle-clonus, more marked on the right side, upon the initial examination.

Memory defects were largely of an extrinsic order, being due rather to inattention and apathy than to demonstrable deterioration. On the other hand, there seems to have been a true deterioration in calculating ability (patient formerly able to make change properly); but how to interpret this is doubtful.

Inattention, apathy, allopsychic (probably autopsychic) delusions, defects of judgment, irritability, impulsive violence, resistiveness, seclusiveness, and postural mannerisms formed a picture possibly suggestive of catatonic dementia præcox, although very probably the onset of the case should be taken to indicate a paranoic condition with some sexual nucleus.

The Wassermann reaction in serum and fluid was negative, and the fluid was cytologically negative.

*Summary of autopsy findings.*—Death was due to *tuberculous pyopneumothorax*, besides which there were epithelioma of tongue, with micro-

scopic pituitary metastasis, and submaxillary metastasis; emaciation and scaphoid abdomen; chronic endo- and myocarditis; chronic peritonitis (liver and spleen); coronary arteriosclerosis, slight; chronic and acute nephritis; mesenteric lymphnoditis.

The brain weighed 1165 gm. According to Tigges' formula, the brain should have weighed 1240 gm. (subject 155 cm. long). *Pial thickening over vertex.* Brain firm. The *superior frontal regions* of both sides show a mild degree of *atrophy* (or *aplasia?*). The mesial surfaces of the pre-frontal areas show somewhat deep hollowing of sulci. Perhaps the *left angular gyrus* region may be thought to show the greatest degree of *atrophy* (or *aplasia?*). The corpus callosum is of proper dimensions and without thinning.

The whole brain gives the impression of a mild atrophic (and proportionately sclerotic) process which has affected the areas above mentioned only a little more than the remainder.

The right temporal area is the simplest in construction found anywhere in the present series, if the sulcus count can be trusted. As in Case XII, there is a degree of internal hydrocephalus, and here still more sharply to the posterior cornua of the lateral ventricles.

A woman with largely paranoid symptoms for six or seven years, and with mental change beginning insidiously at 20. Death at 29. The question between paranoia and catatonia is reflected in the autopsy findings. Both anterior and posterior association areas are affected (superior frontal and angular gyrus). Correlations can hardly be safe, on account of the generalized character of the lesions.

CASE XIV.—N. H. (D. S. H. 16016, Path. 1571) was a case regarded as *paranoid dementia præcox*. Thought to be "going to pieces" at 45 to 46. A "rolling stone," sometimes in fairly good circumstances. Academy student three years (failed to graduate), reporter, book-agent, insurance agent, theatrical work, bicycle business, Spanish-War veteran. Syphilis at 28. Married at 41, later divorced.

Somatic delusions: *scalp tied down, hair growing in.* "*Hair backed up behind my neck as though it was caught behind my ears. Kept growing and growing until it sort of solidified, making one side more noticeable than the other.*" (Thought he resembled Sir Henry Irving, especially with regard to hair.) *Rectum out of line. Whole body drawn toward the left*, so much that patient could "*feel a great flap under left arm.*" Picked at sores (some, results of self-mutilation).

After six months, a certain improvement; ideas about body corrected, except that hair still made him feel uncomfortable, and he still picked at sores on body. Later felt a *tightness of chest*. Death at 54.

*Summary of autopsy findings.*—Cause of death: *Bronchopneumonia.* Acute lesions: Conjunctivitis, otitis media, abrasions.

Chronic lesions: Aortic and coronary arteriosclerosis, mitral and aortic

valvular endocarditis, cirrhosis of liver (1000 gm.; stellate scars), constriction of œsophagus, scars of apices of lungs, subcapsular cysts of kidneys.

Nervous system: The scalp is not unusual. The calvarium is asymmetrical, measuring 3 mm. in the frontal, 3 mm. in the temporal, and 5 mm. in the occipital region. There is a very small amount of diploe. The tables are thin. There is some asymmetry in the floor of the skull. The left frontal fossa seems flatter than the right, the right being more pointed anteriorly and sloping back more acutely than the left. The crista galli is deviated to the right from forward to backward. The left temporal fossa is broader than the right; the bone bulges out more on the left side than on the right. The right occipital fossa appears larger than the left. There is no particular deviation of the sinuses. The lateral sinuses are clear. The right middle ear shows considerable injection, and there is a small amount of serous, possibly purulent, material there. The brain weighs 1490 gm. (over weight by Tigges' formula— $8 \times 165 \text{ cm.} = 1320$ ). The pia is slightly clouded over the sulci and over the vertex. The pial vessels are somewhat injected. There is considerable Pacchionian granulation. The brain is fairly symmetrical, and everywhere firm. The convolutions are well rounded and there is no gaping of the sulci. There is slight flattening of the convolutions in the precentral region on the right side, more than on the left. The temporal lobes are the softest portion.

The pons and cerebellum weigh 175 gm. The ependyma of the fourth ventricle is smooth. The pons and cerebellum are firm.

Paranoid dementia præcox. Delusions about scalp and hair, bodily distortion, possibly due to sensory impulses from a marked cranial distortion or to local cerebral pressure (left temporal lobe accommodated in less space than right, flattening of right precentral region—somatic delusion of *great flap under left arm.*) There were various effects of syphilis (of which there was a history at 28, 26 years before death), such as stellate scars of liver and œsophageal constriction (somatic delusions of *tightness in chest*).

The interpretation of this case would be easier if we could suppose that the skull distortion and brain pressure were responsible for some of the symptoms (compare my paper, "The Somatic Sources of Somatic Delusions").

The sulcal counts of this case indicate the comparative general simplicity of the gyri.

CASE XV.—A. C. (D. S. H. 16669, Path. 1574) was a case observed in hospital less than three months, and in which the diagnosis *dementia præcox* was only a retroactive one. The patient, a well-educated single woman of American stock, was the eldest of five children, all regarded by some as peculiar (one brother unbalanced and unable to take care of himself); and her father was a suicide at 43 in a despondent fit (mother died of cancer).



Patient always an invalid. Scoliosis. Left talipes equinus, shortening and atrophy of left leg, arthritis of left knee with condylar hypertrophy, left knee-jerk sluggish. Flattening of left occipital region of skull was a striking feature during life. Somewhat deaf (purulent otitis media, right, at autopsy).

*Melancholy spells off and on from the 40th year* (poison purchased at times). After 55, conceived exaggerated *ideas of wealth* (gave pipe organs to churches, and handed 20-dollar bills out when her fortune did not permit this). At 59, there was an *appearance of dementia* (unkemptness, untidiness, lack of spontaneous conversation, monologue about fairies) and possibly some actual losses of school knowledge. Restlessness, noisiness, and loquacity preceded her admission to hospital.

Impairment of vision (no arcus senilis). Aside from slight *tremor of tongue*, no neurological disturbance.

Autopsychic delusions: "*Not in human form*," "*I try to make myself into a fairy, but I can't vanish myself*," "*Feels strange*." Sometimes thought people about her were *scarlet fairies*. Said she had a *choking feeling in neck* (cf. *cystic thyroiditis* found at autopsy).

*Summary of autopsy findings.*—The cause of death is doubtful, but the atrophic spleen (16 gm.) and the *atrophic intestinal mucosa* and *enlarged mesenteric lymph-nodes* suggest the terminal process. The brain tissue was a little soft. Acute cell changes in three levels of spinal cord, and diffuse Marchi blackenings. There were also a mild *pneumonic* process in the right lung; emaciation; gastropnoia; chronic aortic endocarditis; slight sclerosis of aorta, common and internal iliac arteries, and basal cerebral arteries; chronic parenchymatous nephritis.

The brain (1235 gm., over weight by about 90 gm. according to Tigges' formula; but the scoliosis had reduced the body length) was of a normal appearance in the gross, save for pial thickening over the vertex. There was a slight *presplenial thinning of the corpus callosum*. The *frontal and parietal opercula* of the left side show a slight retraction indicating a possible slight atrophy. The superior temporal regions are well developed and possess more cross-subdivisions than usual.

The cephalic asymmetry is reflected in the brain in the form of a slight flattening of the left hemisphere as if by a force operating inwards and downwards from the left.

It is not clear that this case can safely be termed one of dementia præcox on the score either of its course or of its terminal phenomena. It may more properly belong in one of the more obscure *presenile* groups, in which a hereditarily tainted person may be predestined to fall victim to a process of unknown character, possibly resembling that of *manic-depressive* insanity.

Case XV was another case (compare Cases V, IX) of diagnosis "*dementia præcox*" retroactively made. There is no evidence

of either hallucinosis or catatonia in the case. It is not certain that the case is one of *dementia præcox* (see above). The delusions of grandeur taking effect in overt extravagant acts and the fantastic autopsychic delusions can be related possibly to some process barely indicated in the gross (frontal and parietal opercular atrophy); it is striking that the temporal lobes look perfectly normal.

CASE XVI.—A. D. (D. S. H. 14453, Path. 1575) was an *unclassified* case, thought to be of the *dementia præcox* group. The patient was of an old New England family and comparatively well-educated (Chautauqua supplement). Hotel housekeeper, matron of orphan asylum, later dressmaker with shop. "Iron-willed." *Always peculiar*, like other members of family (no insanity in direct line). Became interested in *Christian Science* at 42, but ceased to believe latterly. Climacteric stated to be at 56.

First appearance of mental symptoms variously stated from 58 onwards. At 60, had a "*piano which played without hands*." At 59, bought hens to sell eggs, but the *hens became disfigured, humpbacked, human-faced*. An old horse became *hypnotized* and covered with sores in her presence, only later to be restored to normal. Similar ideas about human beings, commingled with *Christian Science* doctrines. Her niece willed her a *cashmere kimona* (actually brought for repairs); and this, on obtaining, she kept.

After a year in hospital, sudden change of disposition: disturbed at night, threw water in patients' beds (symbolic baptisms?), seclusive, inaccessible, violence to patients (striking or kicking at approaching persons with an air of great calmness and quietude).

Physique remained perfect until 65, when an epigastric mass, yellowish pallor of skin, nausea and vomiting appeared; and patient died within a month.

*Summary of autopsy findings.*—Death was due to *general carcinosis* (stomach, pancreas, lungs, peritoneal wall). There was *no emaciation* (thyroid small, soft and flabby, histologically not abnormal with respect to colloid).

Various evidences of chronic disease in trunk: Nephritis, vegetative endocarditis, apical scars of lungs, perisplenitis, myocarditis (edema of ankles) slight aortic and common iliac sclerosis, adhesive pleuritis.

The brain weighed but 1075 gm. and showed *general atrophy*, but perhaps more especially in the post-Rolandic and supra-Sylvian tissues. It is remarkable that *no dilatation of ventricles could be found*. The corpus callosum was of proper proportions. The coronal sections strikingly show the *gaping of sulci in the superior and inferior parietal regions*.

The *gross* distribution of lesions in this brain is suggestive from the standpoint of psychopathological analysis. The imaginative play in the case was unusual, and was commented on early by the

patient herself; and the pseudohallucinational or illusory character of many of the patient's ideas was striking. The increasing and morbid reticence of the patient permitted no analysis of the basis of her catatonic violence ("tranquil" but violent kicking of patients); but remembrance of the *hypnotized horse suddenly broken out with sores* gives a hint of possible mechanism.

It is by no means clear that this case should be dubbed dementia præcox; possibly it belongs in the difficult *presenile* group. Instead of, however, like XV, approaching manic-depressive insanity, the case would appear to approach more a dementia præcox (Christian Science, paranoia, catatonia). Metaphysical delusions (pseudoidealistic conceptions of environment) seem in any event to be associated in this case with parietal-lobe atrophy (little transcortical or subcortical disorder!) which deserves extended histological analysis.

CASE XVII.—E. L. (D. S. H. 16674, Path. 1583) was a case regarded as beyond question a *catatonic* example of *dementia præcox*, but with the unusual complication of *glycosuria* (a brother died of diabetes at 12). The patient was of old Vermont stock, had received a partial education, became a domestic, and married at 20. The first child was healthy, the second had spina bifida, the third was a miscarriage followed by nervousness, emotionality, scolding habits; a month later, glycosuria, visual and auditory hallucinations, delusions of poisoning and of influence from a machine, outbursts of excitement, and refusal of medicine and food developed.

Hair of head thin, silky, infantile-feeling. Eyebrows and lashes thin or absent. Axillary and pubic hair scanty. Trophic disorder of finger-nails and toe-nails. Cyanosis of feet and hands, especially of feet.

Pulse during examination often rose to 174.

*Cereæ flexibilitas* marked. *Paræsthesia* described.

Delusions: *Moles* of abdomen described as injection places made by doctors. "*Throat filled up with lead*," "*Poison in saliva*."

Auditory, tactile and visual hallucinations.

*Cereæ flexibilitas*, impulsive violence, resistivism, refusal to take diabetic diet, furunculosis, finally diphtheria. Death in less than nine months from onset. Wassermann: serum negative.

*Summary of autopsy findings*.—The autopsy showed a *small hard thyroid*, a *large much injected pituitary gland*, a *small, nodular pancreas*. Microscopic study suggested pancreas as possibly related with the glycosuria. Parts of the pancreas showed apparently normal islands of Langerhans, others few or small ones; in some places polynuclear leucocytes had gathered in considerable numbers in the connective tissue adjacent to islands, but were not often found inside. Whether these leucocytes came in response to necrosis is doubtful.

Death was due to *diphtheritic tonsillitis and pharyngitis*. In addition to the dermal anomalies mentioned above, the autopsy showed a *patent foramen ovale, aortic and common iliac sclerosis*, chronic pleuritis, cholelithiasis, and acute pneumonic areas in each lung.

The brain weighed 1130 gm., possibly a loss or absence of over 100 gm. It lay in an asymmetrical skull, in which the right middle fossa was deeper and broader than the left, to accommodate a *right temporal lobe larger than the left* and of a different (more rounded, blunter) shape. This disparity in size of the temporal regions is exhibited in the coronal sections, which show that the hippocampal gyri do not share in the inequality to any appreciable extent and that the *temporal inequality fades out posteriorly*.

There is a striking difference in the general look of the two hemispheres when viewed from the flank, so much that the casual observer might not readily believe that the two views were taken from the same brain. The sulci of the *right hemisphere are more numerous and complex* than those of the *left*. This is everywhere evident, but nowhere more so than in the parietal regions and at the junction of the occipital and temporal regions.

Moderate *presplenial thinning of corpus callosum*. The cranial inequality above mentioned, taken in conjunction with the convolutional inequalities, leads to the hypothesis that this case exhibits many congenital or early brain difficulties, which existed prior to her mental symptoms.

Catatonia and hallucinosis, and also paranoia (early, allopsychic; later somatic) characterized this case. The anomalies seen are largely post-Rolandic and infra-Sylvian.

CASE XVIII.—M. W. (D. S. H. 169, Path. 1593) was a case of so-called "*chronic mania*," which by reason of her development of *cataplexy, cerea flexibilitas* and *neologisms*, might well be regarded as a case of *catatonic dementia præcox*.

*Parents peculiar*. Patient *solitary and peculiar* at 27. Six months in McLean Hospital at 33. *Denudative, untidy, deluded, incoherent*; admitted to D. S. H. at 37. Delusions bore on love; *wanted to kiss every man she met* (39 years). At 43, *violent*, apparently demented. At 46, *fat*, inactive, lazy. At 55, lump appeared under left mastoid muscle and remained for about six months. At 64, *cataplexy, cerea flexibilitas, mutacism*. At 68, delusions of *grandeur* (orders conferred on brother). Neologisms: asked if she slept well, replied, "*Oh! yes, slumosee*." Asked if food was good, replied, "*Yes, except—coplex—my brother—the great order of—except—mia—plumbo*." Death at 72 after ten days fever and vomiting.

*Summary of autopsy findings*.—Death was due probably to septicemia from an ulcerative purulent *cholecystitis* and hypostatic *pneumonia*.

Chronic lesions were: Valvular and parietal *endocarditis, myocarditis, pleuritis, perisplenitis, cholelithiasis, nephritis*, aortic and iliac *sclerosis*, fibromyoma of uterus. The liver was *fatty, ovaries cystic, bone-marrow pale*.

Head: *Right-sided frontal endostosis, adherent dura*. Brain weight 1260 gm. (almost normal according to Tigges' formula, 8 x body length, 160



cm. = 1280). The gross appearances, however, indicated considerable loss of weight, in the shape of a *generalized cortical atrophy* (unless the appearances be taken to indicate aplasia?), especially marked in the supra-Sylvian tissues, *superior frontal*, *precentral*, and *postcentral* areas. The interparietal sulci both anastomose with the Sylvian fissures, and by means of wide bay-like spaces.

It is interesting that the *corpus callosum* is not appreciably thinned out, nor are the ventricles appreciably dilated. The analysis shows that, if we are dealing with an atrophy, it is a rather differential one which does not affect the commissural fibers of the callosum.

Correlations are interfered with by the generalization of the atrophy in this case of 44 years' duration.

CASE XIX.—J. K. (D. S. H. 10037, Path. 1602) was a case of *paranoia*, onset at 34 years. A dressmaker, entered W. S. H. at 34, never well since.

Entered D. S. H. at 50 years. Hears enemies talking. Kept awake nights. Lungs torn out by electricity. Has earned a fortune—\$100,000 in one year, robbed by a gang. Shocks from a dynamo, followed by bad, heavy feelings in limbs. Gang worked a tube on her, blew gas in nose, producing attacks of coughing and sneezing. Applied to police for protection.

At 52 years, double tertian malarial fever.

At 58 years, *machines are used to tear up back and tear head from body*.

At 61, *hips torn to pieces and hair was torn off*. Later, *fingers were pulled apart, as well as joints and eyebrows*.

Onset of thrombosis and enteritis three days before death at 62 years.

Autopsy 28 hours post mortem.

*Summary of autopsy findings*.—Cause of death: *Acute enterocolitis, myocarditis*.

Acute lesions: Bronchopneumonia.

Chronic lesions: Atrophy of intestinal mucosa; aortic sclerosis, internal carotid arteriosclerosis, tricuspid valvular sclerosis; chronic nephritis; chronic splenitis; cholelithiasis; cystic right ovary; favus of vertex; chronic pachymeningitis; arcus senilis; grooving of calvarium for vessels; absence of teeth.

Nervous system: Brain weight 1315 (excess 59 gm. by Tigges' formula, 8 x body length, 157 cm.).

The brain of this case was distinguished by a marked diffuse chronic *pial thickening* (especially over vertex) and by a marked degree of *internal hydrocephalus* (such as would hardly have been suspected from the brain's external aspect or its weight of 1315 gm.). There was only slight basal cerebral arteriosclerosis. The grossly evident *posterior-column degeneration* of the spinal cord might also give rise to suspicions as to the integrity of the brain itself (or even suggest a luetic origin for the total picture). The *posterior half* of the main part of the *corpus callosum* was slightly *thinner* than normal. The frontal polar gyri are richly *subdivided*, especially on the orbital surface (also the *lingual gyri*). A double commissure was discovered on dissection.



Possibly a case of *paraphrenia phantastica* (compare Cases IV, VII). Like IV and VII, XIX shows generalized disease, but of a different genesis (leptomeningitis and internal hydrocephalus).

CASE XX.—Z. T. (D. S. H. 6334, Path. 1603) was a case of "secondary dementia" with symptoms dating back to 33 years or earlier. At first, paranoïdal (homicidal, suicidal, "poisoned") and unusually dangerous. In hospital, given to impulsive violence—knocked down and stamped upon a patient because "he would not dress and had over some of his talk to him."

Imagination unusually varied: expansive ideas about wealth, mines owned, relation to royalty. Would often state that he was some notable person. *Had a job polishing off the moon. Moon floats in the ocean like a bubble in the day, reached by man-of-war.* Drew "*marine views*" with colored water got by soaking colored paper. "*Bushes growing inside of body,*" "*daughter inside,*" "*1000 years old.*"

Glycosuria at 50. Death at 67.

Autopsy 22 hours post mortem.

*Summary of autopsy findings.*—Cause of death: *Cerebral hemorrhage.*

Acute lesions: Acute splenitis, acute bronchitis.

Chronic lesions: Obesity; coronary, internal mammary, and cerebral arteriosclerosis; mitral and aortic valvular endocarditis; chronic nephritis; fat-replacement in pancreas; arcus senilis; chronic thyroiditis.

Brain weight (with hemorrhage) 1550 gm.

Death in this case was due to an extensive *right-sided cerebral hemorrhage*. (Cf. Case XXII (1616).) It is clear, however, that a marked *chronic pial thickening over the vertex* and a marked degree of *internal hydrocephalus* antedated the hemorrhage.

Perhaps the most striking feature of the brain is an anomaly of the *left superior temporal gyrus* remarkably similar to that of Case III (1319). It is interesting that the coat of pial thickening spreads down from the vertex *over the left opercular region* (not markedly on the right) and is especially marked *over the left frontal operculum* (where stripping off the membrane reveals a somewhat deep-lying gyrus, perhaps presenting a similar type of anomaly to that of the above-mentioned superior temporal anomaly).

Investigation of this case histologically might seem unprofitable were it not for the somewhat similar anomaly of Case III (1319), in which hemorrhage did *not* occur and in which the age at death was 31 instead of 60. It will be noted, however, that there was also in Case III (1319) an internal hydrocephalus, but more marked on the left side.

This case, like XV, seems to show little evidence of either catatonia or hallucinosis, and is marked by delusions of grandeur and fantastic autopsychic delusions. The brain shows frontal opercular, and to some extent parietal opercular, lesions; but,

unlike XV, this case shows anomaly of superior temporal convolution.

CASE XXI.—A. G. (D. S. H. 14001, Path. 1615) was a lady's maid, born in France, queer after 34 years of age. Ideas of reference (auditory hallucinosis) at 45. Strong feelings in forehead (a girl had put chloroform there, she said). *Chloroform and powders smelled. Voices* ("bad woman," etc.) *heard*. Hyperreligiosity. Wrote innumerable prayers in archaic French and bound them in books of her own manufacture. "*Absences of thought*." One seemingly hysterical attack, while menstruating, followed by hypalgia, incontinence of urine, muscular weakness, and later by amnesia.

*Summary of autopsy findings.*—Cause of death: *Laryngeal diphtheria*.

Acute lesions: Dermatitis of face, hands, wrists, genitalia.

Chronic lesions: Periappendicitis and generalized adhesive peritonitis; obliterative pleuritis, right; cholelithiasis.

Nervous system: Brain weight 1170 gm. (8 x body length, 140 cm., would equal 1120).

Attention is at once drawn in this case to the two *inferior parietal regions*, which are both (and more notably the left) subject to a kind of general retraction from the main contours of the surrounding parts. This retraction appears to center about the *interparietal sulci* in their *posterior* portions. Over these sulci and the inferior parietal lobules there is a slight *pial opacity*, without demonstrable thickening and with borders playing off vaguely into the adjacent normal-looking pia mater.

There is a marked rather suddenly appearing *thin place* in the posterior quarter of the main part of the *corpus callosum*.

The impression is also conveyed of a slight to moderate degree of *atrophy of the opercular region*, more marked on the left side. Internal hydrocephalus, if present, is but slight in amount.

Hallucinosis seemed to command the paranoia of this case, and it is a question whether the long archaic French prayers were not dictated by voices. The paranoia seems on the whole "impressional" or ideomotor, and therefore consistent with the (inferior) parietal and opercular atrophy.

CASE XXII.—E. A. (D. S. H. 1227, 2009, Path. 1616) gave first symptoms at 26: melancholia, after business reverses; fainting fits; idea of servant girl as a Boston lady in disguise (attempted to enter her room on the ground that he was married to her). Improved somewhat in hospital, but returned at 27. At 29 spoke of God's commands. Assumed a German accent. Occasional violence, surliness, abusiveness. At 33, muttering and gesticulating. At 35, rhythmical motions with one hand. At 45, walked with head bent back and turned to one side. Mutism for years. Shock at 56 (transient spastic right-sided paralysis, clonus, Babinski). A diagnosis of organic dementia (luetic) was then considered: After being mute for years, patient suddenly spoke, objecting to a throat culture being taken. Death at 59.

*Summary of autopsy findings.*—Cause of death: *Cerebral hemorrhage*.

Acute lesions: Hemorrhages in floor of fourth ventricle, pancreas, stomach, right sclera; early ulceration of colon; large mesenteric lymph-node; bronchopneumonia.

Chronic lesions: Aortic sclerosis; pleuritis, right side; periappendicitis; pigmented moles.

Nervous system: Brain weight 1220 gm. (8x body length, 164 cm. = 1312), a reduction despite the hemorrhage of the right hemisphere.

Like Case XX (1603), Case XXII (1616) died of *right-sided cerebral hemorrhage*. Like 1603 also, the case showed *internal hydrocephalus*. There was, however, no marked degree of pial thickening.

Curiously enough, there is also in this case an approach to the *left-sided superior temporal anomaly* shown in Cases II (1317), III (1319), XX (1603). This case appears to suggest a vascular origin for the anomaly, in that numerous apparently serpentine vessels were found coursing over the opercular regions.

CASE XXIII.—L. S. (D. S. H. 11176, Path. 1622) was a case of *dementia praecox*. Onset at 22 years. Entered McLean Hospital at 22. Love affair. Told she was making herself and family conspicuous, wept all night. Moody, praying, said strange things, taciturn, apathetic, suicidal threats. Heard father calling her. Disoriented. Violent and profane. Occasionally laughed and sang. Muscular spasms and clonic spasms when asleep.

Admitted to D. S. H. at 24 years. Oriented. Depressed. "Its a pity she is so wicked." "My body is vile." "Tormented with voices, ashamed of everything." Insight. Violent outbreaks, ideas of persecution and unreality. Somatic ideas (*head and brain of sawdust*). Lay about on couch, hair down. At 29 to 30 years, mute and resistive, constantly hallucinated, "men in her room to lead her astray," oriented, unsocial. Later denudative. Four days before death at 34 years, developed lobar pneumonia.

*Summary of autopsy findings.*—The brain of this case has a simple pattern. (Compare, e. g., the richness of sulcation in the *orbital and lingual gyri* of Case XIX (1602).) The *parieto-occipital fissures* are deeply sculptured. The sulcus which penetrates the *left supramarginal gyrus* seems also unduly deep. The *sulci separating postcentral gyri from inferior parietal lobules* are continued into the fissure of Sylvius, forming a kind of ascending middle branch to each; these are also *deeply hollowed out*, as also are the ascending branches of the Sylvian fissures. There is a slight chronic *leptomeningitis*, most marked over the mouth of the *left Sylvian fissure*. However, one gets the impression that the *superior parietal atrophy* (or *aplasia?*), as represented by the deepening of the parieto-occipital fissures, is the most marked lesion of the brain.

The corpus callosum strikes one as of good proportions, and *fails to show thinning out posteriorly*.

At first paranoid, later catatonic, this case offers difficulties in correlation. Hallucinosis was prominent at various periods in the course, but there is little to show in the gross brain. Therefore, since the very pronounced atrophy is mainly above the fissure of Sylvius (microscopic examination, it is true, shows considerable change in the superior temporal gyrus, but the left supramarginal gyrus leads all other areas in extent of atrophy from the microscopic point of view), we may need to be content, so far as gross correlations go, with a parietal correlation for the catatonia.

CASE XXIV.—L. L. (D. S. H. 16063, Path. 1625) had a paternal uncle insane (type unclassified), and was always seclusive. Married at 22 (four children), lost husband at 30, worked hard. At 34 was unconscious from a fall, and four months later became strange and talked strangely (ideas of reference; disconnected letters; catatonic stupor 10 days, two days lucid, again stuporous or catatonic till death at 39). Denudative, resistive, negativistic, verbigeration, untidiness, assumption of fixed positions, at times cerea flexibilitas, silent with doctors.

*Summary of autopsy findings.*—Cause of death not clear.

Acute lesions: Cervicitis, injection of ileum.

Chronic lesions: Emaciation; large mesenteric lymph-nodes; chronic nephritis, with focal areas of sclerosis; gastroptosis; bones brittle.

Nervous system: Brain weight 1190 gm. (1200 gm. by Tigges' formula). Slight leptomenigitis along sulci.

The most striking feature of the brain of this case is the somewhat sharply marked *thinning of the posterior half of the main portion of the corpus callosum*. There is also a moderate *dilatation of the posterior halves of the bodies of the lateral ventricles*.

The *convolutional pattern* of the brain gives the impression of *simplicity*, except upon the inner face of the occipital regions, which is either atrophic or aplastic, judging from the gross appearances.

Especially simple in construction appears to be the temporal lobe of each side. There is another example of anomalous Sylvian branching, reminding one, more especially on the left side, of conditions in Case XXIII (1622). The area of Broca and the co-ordinate right-sided area show rather deeply hollowed sulci.

This almost exclusively catatonic case is of note in that all the pre-Rolandic tissues fail to show appreciable lesion, whereas a somewhat marked posterior dilatation of the ventricles, thinning of the posterior part of the corpus callosum, and occipital atrophy or aplasia (inner surfaces) and simplicity of the temporal lobes emphasize the sensorial and supersensorial nature of the lesions. (Microscopically, all areas examined showed marked satellitosis.)

CASE XXV.—L. P. (D. S. H. 16779, Path. 1634) was a case of *dementia præcox*, onset at 17 years. Father insane, *dementia præcox*. At 17 *feared Catholics. Men creeping in through window, devils and negroes*. Ideas of persecution.

Admitted to D. S. H. at 17 years. Restless, talking incoherently and impulsively singing. Motions quick and impulsive. Mannerisms, tube-fed, untidy.

*Cereæ flexibilitas* 18 days after entrance. Hums to herself, recognizes mother but does not talk to her.

Symptoms thought to be those of pulmonary tuberculosis two months before death at 18.

*Summary of autopsy findings*.—Cause of death: *Tuberculous abscesses of left lung*.

Chronic lesions: Chronic obliterative pleuritis, left.

Brain weight 1270 gm. (excess 142 gm. by Tigges' formula, 8 x body length 141 cm.).

This brief case of catatonia, having a brain practically normal in gross (with a suggestion of ventricular dilatation posteriorly), was studied microscopically in advance of the total series, and shows marked characteristic changes everywhere. Correlations of lesions to particular symptoms are accordingly impossible.

Perhaps this is a case in which tuberculosis has something to do with the process. At least the tuberculosis and the mental process appear to have run their course together.

#### V. GENERAL DISCUSSION.

I am disposed to believe that the present work, confirmatory as it in general is of my former work of 1910, goes very far toward placing *dementia præcox* in the structural group of mental diseases. Previous work of several authors had left much to be desired in respect to proofs of the structurality of this disease, because reliance was placed on *microscopic* examination and the observation of various cell changes, some of which might well be reversible or terminal or intercurrent, or even agonal. Even the initial conclusions of Alzheimer concerning deep-layer cortical gliosis in catatonia (1897) are now replaced by Kraepelin (1913) with an emphasis on changes in *supra-stellate* layers. Nor did any author seem to reckon with the fact that such microscopic changes as are sometimes adequately described must inevitably leave, as a rule, some macroscopic trace, if the disease had lasted a long enough time (say three months, more or less). Accordingly, in



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the absence from the literature of all systematic gross brain study in dementia præcox, at least of such study in any large series of cases, I undertook to fill the gap and have fortunately been able, through grants from the Massachusetts State Board of Insanity, to execute the work with the aid of systematic brain photography. Careful and systematic autopsy protocols, brain photographs systematically taken, and microscopic sections in all cases of possible doubt as to gross diagnosis, have formed the basis of the work on the structural side.

Before considering the data, it may be inquired whether the cases presented are really cases of dementia præcox, since *diagnosis* in this disease or disease group is admittedly difficult. The condensed histories given in the text speak for themselves. Possibly some cases of manic-depressive insanity of mixed type may have crept into the series; if so, the anatomical correlations could only be lowered by our diagnostic error. There may be instances of imbecility in the series, but none, I believe, without a strong dementia præcox coloring; if such diagnostic errors were plentiful, it is likely that the correlation with agenesic lesions would become too high, and the hypothesis of acquired hypoplasia (otherwise acceptable) would correspondingly suffer. In any event, I believe, there is no false bolstering of the high anatomical correlation by means of cases having acquired lesions obviously too recent or otherwise theoretically irrelevant to explain the symptoms.

*Among the 25 cases systematically studied, there were but two without evidence of cortical atrophy.* These two (VIII, of three weeks' duration, and XXV, of two months' duration) may not have been cases of dementia præcox (since it is doubtful whether the diagnosis can be made safely in so brief a period); but on the whole it seems desirable to count them as such. One of them (VIII) had a decidedly asymmetrical face and brain (as well as other somatic anomalies). Both cases showed microscopic changes adequate to account for many symptoms. Indeed, the universality of microscopic changes in Case XXV would lead to the idea that this case was proceeding towards a general mild atrophy (should survival have permitted). The brief duration of these cases probably accounts for the absence of macroscopic evidence of brain disease.

What is the *nature of the lesions* found? It seems that they

might be (a) *acquired* by the individual or (b) *congenital*; and evidence in each of these directions had been supplied from the work of 1910. But, if acquired, the lesions may still have arisen quite out of relation to the development of symptoms. Thus, they may have been *acquired early* in life and thus have been tantamount to congenital lesions, or at all events extinct so far as symptom-production is concerned. Or, on the other hand, they might come to be *acquired late* in the course of the disease, and be either chronologically *secondary* to the disease or even *incidental* and adventitious results of something quite apart from the disease. If *congenital*, the lesions might be examples of *agenesia* in the sense of a circumstance under which certain cells altogether fail to be developed, or they might be examples of *aplasia or hypoplasia* in the sense of a failure of cells which have been quite properly laid down to progress in the normal direction, *e. g.*, to the normal size, or with all the normal appendages, or with normal powers of resistance, or the like. Besides these possibilities, conditions like *hydrocephalus* might occur (see below), in which anomalous results would follow upon cell readjustments entailing neither *agenesia* nor *aplasia*. And, again, it would be conceivable that *acquired skull distortion*, *abnormal cerebrospinal-fluid pressure*, *abnormal blood pressure*, and cognate conditions (such as have been recently discussed by my colleague, H. M. Adler), should bring about abnormal conditions without the loss of a cell, at least in early phases of the particular abnormal physical state. And combinations of such acquired and congenital features might occur, and some of them were previously noted to occur (1910).

Whatever the general outcome of the inquiry, it might well be that *loss of brain weight* should eventually supervene. In 1910 I found a slight loss of two per cent; the present series shows such marked variations from case to case that conclusions must be withheld, although there are striking individual instances of loss.

Heart, liver, and kidneys show proportionally more loss of weight than does the brain. The kidneys are more atrophic in this series than in the series of 1910. Excluding certain cases with long-standing splenomegaly, the spleen also shows a tendency to low weight.

*How far are the lesions and anomalies of this series interpretable on the hypothesis of dementia præcox as incidentally organic?*



In the first place, the lesions and anomalies are practically constant in occurrence in the new series (23 out of 25 cases), and there is important evidence of similar focal lesions in the great majority of all cases so far examined (45 out of 50 cases). We are left with a small residuum of cases in which no registration in the *gross brain* has occurred. Microscopic examination, so far as we can rely on it, indicates a tendency to similar changes in several of the cases in the "normal-looking" residuum. When we remember the elusive nature of many important gross lesions and anomalies (so often missed in routine autopsies), when we bear in mind that the patient must survive a certain period in order to yield gross evidence of brain disorder at all, and above all when we remember the difficulties in diagnosis in this group, we cannot be surprised at our not forthwith arriving at 100 per cent of gross lesions and anomalies in this series of cases. (As to diagnosis, it should be added that all cases have been included which could by any possibility have been regarded as instances of dementia præcox, and it is safe to say that the percentage of gross lesions has not been artificially increased by any form of exclusion of cases. In particular, I have *not* omitted those difficult cases in which discussion at present rages concerning the differential diagnosis of dementia præcox and manic-depressive insanity of *mixed type*.)

But, though it be granted that our findings are practically constant, it might still be that they are only incidental, much as broken glass might be constantly or almost constantly found after a banquet with wine.

Logical formulæ do not provide an answer to this question. In the particular form in which Meyer has expressed the notion of "incidentally" organic lesions, one sees a suggestion of Edinger's *Ersatztheorie*. Cells, working in such and such abnormal ways, are eventually subject to metabolic changes and final breakdown. Supposing this to be true, and supposing the process to be initiated by some non-cellular event (*i. e.*, by some event that does not interfere at once with normal cell-functioning), still the progress of cell-functioning is by hypothesis sooner or later disturbed. Thus, if I am correct, the disease might be regarded as having two phases—a non-cell-injuring phase and a cell-injuring phase.

I am far from certain that this issue can now be resolved, and I see the tactical advantage of holding to a non-cell-injuring phase



in dementia præcox (*therapeutic optimism!*). But, if the issue is constantly or even very frequently a phase of cell-injury, then it is plain that something of peculiar potency must lurk in the non-cellular events that initiate dementia præcox; and, what is still more important, it is obvious that the cell-injuries themselves must have an important bearing on the progress of the disease. Thus I am loth to admit that there can be "incidental" changes in the brain which will not be of profound significance to its function. The brain is so labile an organ, and many parts of it phylogenetically so new, that even small changes must be of great consequence to it.

Accordingly, in exchange for the tactical advantage of a provisional therapeutic optimism, I would prefer the strategic advantage of a research point of view which should study intently the cellular results (if they are results), the cellular progress, and the cellular causes (if there are any) of this disease.

As a last word concerning this problem, I would say that I think there are reasons for supposing that there may be no fundamental difference between the so-called structural ("organic") and the so-called functional points of view. I believe that some day it may transpire that events apparently non-cellular are really cellular, or, otherwise expressed, there may be but one series of events, some part of which is conveniently termed psychical and some part physical. This is no place in which to discuss this view; I need only say, in addition to already published remarks, that the new view will probably not serve either to identify the psychical with the physical or *vice versa*.

Granting that a new ontology on such lines should enter to replace the parallelistic or interactionistic ontologies which now prevail, we should be forced, however, not to regard the changes we find *sub specie structuræ* as either causative of or incidental to the changes we find *sub specie functionis*. All relevant and pertinent changes would, to the new point of view, assume their proper place in some higher genus of things and events.

I wish to lay no particular stress on these ontological points here, and merely submit, concerning the concept of the "incidentally organic," that "*incidental*" should not receive the connotation of (a) *infrequent*, since the changes are certainly not infrequent, or (b) *unimportant*, since, after they occur, it is hard to

see how such changes could fail to be of importance. It is possible that the term "incidental" is intended by Meyer to connote a *secondary* nature for these changes; if the changes are secondary but constant, it would be hard to deny them a measure of importance. Possibly, however, "incidental" is meant to be taken as *secondary in point of time* (this is suggested by Meyer's term "endogenous break in compensation"), and this meaning is the one which I should take most seriously. Here would lodge the most momentous issue as to facts. *Does the process of dementia præcox begin with a non-cell-injuring phase?* That seems to me to be the best-defined issue of Meyer's attempt to regard changes found in dementia præcox brains as incidental.

This leads to a consideration of what I understand to be August Hoch's attitude to the anatomical findings in dementia præcox. I understand him to believe that they represent either an agenesis or the effects of such agenesis of the brain. Of course the omitted (agenesic) elements are few, and the resemblance of dementia præcox to higher grades of imbecility is often more apparent than real.

I myself hold rather that such findings as we now have indicating maldevelopment (and there are surely many such) indicate rather an aplasia than an agenesis, rather an interference with development of cells laid down than an interference with their laying down. But, of course, an early aplasia may go far to simulate an actual agenesis, so that the issue is in a particular case often never to be resolved.

In the 1910 series, I studied from this particular point of view 15 cases, of which 14 yielded features which I regarded as "acquired" (the 15th case happens to figure also as Case IV (1335) of this series, and suggests on further study also an acquired atrophy of the occipital, but especially of the temporal, regions). But at the same time, 8 of the 15 cases had more or less pronounced features indicating early hypoplasia or (possibly in some cases) agenesis.

The comparative infrequency (8:15) of these developmental changes might lead one to underestimate their value as an index, and they might be dismissed in one of the senses of the term "incidental." But I am inclined to hold that further study will increase rather than diminish their number.

The following table divides the pertinent lesions found into provisional groups as acquired and developmental. For the purposes of the present discussion, I have pushed into the developmental column all cases that could by any interpretation belong there from the anatomical point of view. I have, for example, put all the examples of chronic internal hydrocephalus in that group, although I do not feel that they all belong there. It is also doubtful whether all the cranial asymmetries are developmental.

TABLE XI.

## ACQUIRED AND DEVELOPMENTAL LESIONS.

NO.	ACQUIRED.	DEVELOPMENTAL.
I (1297).	Left cerebral atrophy.	
II (1317).	Left Sylvian atrophy.	Left superior frontal hypoplasia; hippocampal hypoplasia?
III (1319).	Slight basal leptomeningitis.	Left temporal hypoplasia, especially superior temporal.
IV (1335).	Temporal and occipital atrophy.	Microcardia, microhepar, small brain.
V (1383).	Cerebral atrophy, especially pre-Rolandic, right. Thinning of corpus callosum.	
VI (1413).	Frontal atrophy (slight) (see opposite also).	Callosomarginal anomalous plication (but early encephalitis?).
VII (1472).	Cerebral atrophy, or...	Cerebral hypoplasia (agenesia?).
VIII (1487).		Patent foramen ovale, persistent thymus, facial asymmetry, asymmetry of brain.
IX (1491).	Paracentral atrophy?	Chronic internal hydrocephalus.
X (1507).	(Brain metastasis of carcinoma) frontal atrophy?	Bilateral temporal agenesis (undue simplicity of plication).
XI (1509).		Patent foramen ovale, facial asymmetry, fetal convolutions of spleen; moderate chronic internal hydrocephalus.

NO.	ACQUIRED.	DEVELOPMENTAL.
XII (1518).	Sclerosis of right callosomarginal gyrus.	Probably the lesion opposite is in some sense hypoplastic; small right cornu ammonis; chronic internal hydrocephalus; left frontal aplasia.
XIII (1540).	Superior frontal atrophy (aplasia?) left angular atrophy.	
XIV (1571).		Cranial asymmetry.
XV (1574).	Atrophy of frontal and parietal operculum; thinning of corpus callosum.	Cranial and cerebral asymmetry.
XVI (1575).	General cortical atrophy, especially post-Rolandic and supra-Sylvian.	
XVII (1583).		Marked cerebral asymmetry of gyri.
XVIII (1593).	General cortical atrophy; especially supra-Sylvian.	
XIX (1602).	Marked chronic leptomeningitis.	Chronic internal hydrocephalus (acquired?).
XX (1603).	Frontal and parietal opercular atrophy; chronic leptomeningitis.	Chronic internal hydrocephalus; anomaly of left superior temporal gyrus.
XXI (1615).	Opercular atrophy, more marked, left.	Retraction of both inferior parietal regions.
XXII (1616).		Chronic internal hydrocephalus; left superior temporal anomaly.
XXIII (1622).	Chronic leptomeningitis.	Superior parietal aplasia (atrophy?).
XXIV (1625).	Thinning of corpus callosum.	Partial chronic internal hydrocephalus.
XXV.	Negative in gross.	Negative in gross.

The result of this tabulation is to show that 19 of the 25 cases might possibly be regarded as in some sense maldevelopmental. An impartial witness to whom I submitted the data thought that surely 14 of these 19 were true instances of maldevelopment. If the eight cases of chronic internal hydrocephalus are all acquired and not developmental, then we still have outstanding 11 cases of maldevelopment.

CLINICAL CORRELATIONS WITH CHRONIC INTERNAL  
HYDROCEPHALUS.

The fact that almost one in three of our random series of dementia præcox brains exhibited more or less well marked partial or general chronic internal hydrocephalus, forms a novel and many-sided problem.

The list is as follows:

TABLE XII.

## CORRELATIONS WITH INTERNAL HYDROCEPHALUS.

No.	Sex.	Age.	Classification (Kraepelin, 1899).	Duration.	Nature and degree of hydro- cephalus.
III (1319).	F.	31	Paranoia (imbecile?).	8 yrs.	Moderate, more of left lateral ventricle.
V (1383).	F.	62	Catatonic.	Years.	Marked, especially in mid portion.
IX (1491).	F.	53	Catatonic.	Years.	Moderate, equal on both sides.
XI (1509).	M.	58	Catatonic.	37 yrs.	Moderate, equal on both sides.
XII (1518).	F.	45	Catatonic.	29 yrs.	Slight general, more marked in two poste- rior cornua.
XIX (1602).	F.	62	Paranoic.	28 yrs.	Marked, equal on both sides.
XX (1603).	M.	67?	Paranoic.	34 yrs.	Marked, probably equa (marked by complicat- ing hemorrhage).
XXII (1616).	M.	49	Catatonic.	23 yrs.	Moderate, equal on both sides.
XXIV (1625).	F.	39	Catatonic.	5 yrs.	Moderate of posterior halves of bodies of lat- eral ventricles.

The meaning of this hydrocephalus may be taken up from various points of view.

*Age* is not a specially powerful factor. Four of the cases were under 50 at death, five over 50.

*Duration of symptoms* is suggestively long. The briefest case had symptoms for five years (one is of unknown duration, probably decades). No case of brief duration exhibits hydrocephalus.



On the other hand there are fairly numerous cases of great duration without hydrocephalus. Examples are:

No.	Sex.	Age.	Classification.	Duration.	Area involved.
*I (1297).	F.	47	Paranoic.	27 yrs.	Atrophy confined to cortex.
II (1317).	F.	44	Catatonic.	21 yrs.	Frontal and temporal hypoplasia.
VII (1472).	F.	55	Paranoic.	27 yrs.	General brain atrophy (or aplasia).
X (1507).	F.	60	Paranoic.	36 yrs.	Frontal and temporal atrophy.
XVIII (1593).	F.	71	Catatonic.	44 yrs.	General cortical atrophy
XXI (1615).	F.	58	Paranoic.	24 yrs.	Inferior parietal atrophy (aplasia?).

Thus there are six cases of long duration (over 20 years from first recorded symptoms) which did *not* show hydrocephalus. IV (1335), XV (1574), and XXIII (1622) are three others of fairly long duration (20 or 14 years, decades? and 12 years respectively) which also did *not* show hydrocephalus.

It is obvious, accordingly, that neither the age of the patient nor the duration of the symptoms possesses any regular relation to the appearance of hydrocephalus. It is true that the two most pronounced instances were in the oldest cases—XIX (1602) and XX (1603)—and in cases of long duration (28 years and 34 years respectively). On the other hand, two cases of *shortest* duration—XXIV (1625) (5 years) and III (1319) (8 years)—also showed moderate hydrocephalus, albeit partial (more on left side and in posterior halves of bodies of lateral ventricles respectively). But in one case of 29 years' duration, with death at 45—XII (1518)—the hydrocephalus was also more marked in the two posterior cornua.

It remains to be inquired whether the symptoms of the hydrocephalus cases presented anything in common. The group is in point of fact an uncommonly catatonic group. Although three cases are tabulated as paranoic, in point of fact two of these showed various catatonic or allied features (III, attitudes, resistivism,

\* This case was variable, cyclothymic, and it is a question how much of the period of 27 years was occupied by processes which could affect the brain.

refusal of food; XX, impulsive violence), and the third (XIX) had a great variety of somatic delusions which might well indicate effects wrought in the postcentral gyrus as well as elsewhere. But whether the hyperphantastic somatic delusions of Case XIX are to be regarded as in some sense a catatonic equivalent (heavy feelings in limbs, various delusions of being rent apart), the other cases are all more or less pronouncedly catatonic.

But the group is also somewhat given to hallucinations (III, visual, gustatory (olfactory?); XI, auditory; XIX, auditory; XX, poison (hallucinatory?); XXII, auditory). The cases without hallucinosis were as follows: V and IX, decidedly inadequate history (ideas of poisoning); XII, slight general hydrocephalus, but more marked in posterior cornua; and XXIV, moderate hydrocephalus, confined to posterior halves of bodies of both lateral ventricles. The hallucinatory tendency is therefore stronger when the hydrocephalus is marked and general.

The hydrocephalic group appears not to be so much given to delusions as to catatonia or even to hallucinosis. However, there were three cases tabulated above as paranoic (one of these, Case III, on rather insufficient grounds). Of these three, two were cases in which hyperphantastic delusions, often somatic in content, occurred. It would be my theoretical desire to correlate such hyperphantasia on general grounds rather with lesions or conditions of the posterior association center than with such in the anterior center. There seems to be in these cases of hyperphantasia no necessary development of any morbid or unusual reaction to the delusions expressed. The victim of hyperphantasia is often in a state of passivity like a spectator at the play, and of such a patient it can hardly be said that his attitude or conduct (which we are here attempting to correlate with frontal conditions) is in any respect abnormal.

On the whole, therefore, if I interpret along my previously suggested lines, I see far less evidence of morbid frontal-lobe action (motivated attitude and conduct) than of morbid parieto-temporal action (catatonia and hallucinosis). It is interesting in this regard that there were two instances of hydrocephalus of a more posterior locus, from which it might be thought that for some reason the dilatation is apt to begin there as at a point of lesser resistance to expansion. Both of these local posterior

hydrocephalus cases were markedly catatonic, and neither showed either hallucinosis or delusions (except that XXIV had some ideas of reference).

This problem of the comparative immunity of the hydrocephalus cases from delusions can also be approached from the anatomical side (if the frontal-paranoia correlation be for the moment taken for granted) by inquiring what effects have been wrought by the hydrocephalus cases upon the frontal lobes. These correlations may be studied in detail in the table of Gross Anatomical Correlations with Paranoia (Insane Delusions).

No effects of a gross anatomical nature in the frontal region, whether caused by the hydrocephalic process or not, are to be seen in III, IX (except a slight mesial thinning posteriorly), XIX, XX, XXII, XXIV (except a question of atrophy or aplasia of *inferior* frontal gyri). Of the three cases which do show frontal-lobe lesions, V (moderate frontal atrophy) and XII (left-sided frontal aplasia) had also been inferred in two instances, on clinical grounds, to have shown delusions; whereas in the third (XI), history is lacking. On the whole, therefore, it seems safe to say that, in this group at least, hydrocephalus bears far less hard on the frontal association centers than on the parietotemporal association centers.

Accordingly, if our general contentions be at all correct, the hydrocephalic cases of dementia præcox will be found more often catatonic or hallucinated than deluded (except that hyperphantasia occurs with hydrocephalus).

I assume that, whatever be the conditions provocative of hydrocephalus, it is likely that the process will vary in degree from time to time. It is quite consistent with this conception that the cases of this group should show themselves (as an inspection of the histories will prove) more than usually active and mutable. I also find a tendency to attacks and exacerbations marked in three cases (XI, six attacks; XII, four or more attacks or exacerbations; XXI) and indicated in others.

The first hypothesis which the anatomist would have concerning the cause of the hydrocephalus would be that it is a hydrocephalus due to brain atrophy.

The weights were:

- III. 1250 gm.
- V. 1060 gm. (60 minus, Tigges).
- IX. 1330 gm. (50 plus, Tigges).
- XI. 1265 gm. (100 minus, Tigges).
- XII. 1385 gm. (105 plus, Tigges).
- XIX. 1315 gm. (60 plus, Tigges).
- XX. 1550 gm. (with hemorrhage).
- XXII. 1220 gm. (92 minus, Tigges).
- XXIV. 1190 gm. (10 minus, Tigges).

There is on the face of these figures no convincing evidence of brain atrophy in these hydrocephalic cases, since there is no case which by Tigges' formula has lost more than 100 gm. However, the technical fact must be remembered that the dilated ventricles contain more fluid than normal ones and that, if the brain is weighed at once without thorough drainage (as I am sure has happened at times), the scales may register 100 to 150 gm. of fluid additional to the normal amount (sometimes set at 125 gm.) and thus give a deceptively high value to the brain weight. Accordingly there may be more atrophy than one would suppose from the figures.

In the above remarks I have proceeded as if it were quite likely that the hydrocephalus is acquired. If congenital, we might expect to find various other evidences of aplasia. Case III indicates such aplasia of the left temporal region, probably closely associated with the more marked left-sided internal hydrocephalus. Case IX showed a striking difference in general shape of the two hemispheres (cranial malformation or alteration?). Case XII showed what I have termed above "a right-sided hippocampal aplasia in relation to a left-sided frontal aplasia" (there were also appearances in this case consistent with the hypothesis of an early encephalitis). On the whole, therefore, it would seem that there is not much evidence of an association of the hydrocephalus with congenital features.

#### CORRELATIONS OF TEMPORAL-LOBE LESIONS WITH AUDITORY HALLUCINOSIS.

Following is a table designed to bring out sharply the presence or absence of anatomical correlations with hallucinations of various types (although mainly auditory). The table presents all 25 cases, including 13 cases (IV, V, VIII, IX, X, XII, XIV, XV,

XVI, XVIII, XX, XXIV, XXV) *without* history of hallucinations. In the anatomical column (on the right) have been placed, for these 13 supposedly negative cases, all lesions which might have been thought able to favor hallucinosis. The table can therefore be used in two ways, to try the strength of (a) the correlation between hallucinosis and appropriate cortex lesions (*e. g.*, auditory hallucinosis against temporal-lobe lesions), and (b) the correlation between such lesions and hallucinosis.

The results of the analysis are summed up *infra*.

TABLE XIII.

## GROSS ANATOMICAL CORRELATIONS WITH HALLUCINOSIS.

NO.	SYMPTOMS.	ANATOMICAL CORRELATIONS.
I (1297).	Auditory (reviling, "not real," also noises in head).	Peripheral deafness, bilateral; area of softening, right second temporal gyrus.
II (1317).	Auditory.	Anomaly (with atrophy? or aplasia?) of left superior temporal gyrus. Left temporal lobe poorly supplied with white matter.
III (1319).	Visual?, gustatory?, auditory?	Chronic internal hydrocephalus, left superior temporal anomaly, and poor supply of white matter to left temporal lobe (cf. II).
IV (1335).	<i>No evidence of hallucinations; fantastic delusions (sensorial in origin?).</i>	General hypoplasia (with atrophy?), more marked behind Rolando and below Sylvius.
V (1383).	<i>No evidence of hallucinations.</i>	Temporal and parietal (except postcentral) regions not especially involved. Chronic internal hydrocephalus, especially of mid portion.
VI (1413).	One episode of auditory hallucinations; none later.	No evidence of gross temporal-lobe lesions.
VII (1472).	Early brief period of possibly auditory hallucinations, or possibly ideas of reference construed as hallucinations; later probably <i>Gedankenlautwerden</i> .	General hypoplasia, or more probably atrophy of brain; gyri on both sides of Sylvius slightly more atrophic (hypoplastic) than other gyri.



NO.	SYMPTOMS.	ANATOMICAL CORRELATIONS.
VIII (1487).	<i>No evidence of hallucinations</i> (total course three weeks).	No sclerosis or atrophy elsewhere. Asymmetry of brain (three weeks' duration).
IX (1491).	<i>No evidence of hallucinations.</i>	No sclerosis or atrophy in appropriate regions (paracentral atrophy (aplasia?)). Chronic internal hydrocephalus.
X (1507).	<i>No evidence of hallucinations.</i>	Temporal regions simple in plication; moderate atrophy of temporal lobes.
XI (1509).	Auditory hallucinations inferred from some of patient's attitudes.	Atrophy or aplasia of anterior portion of each superior temporal gyrus. Moderate chronic internal hydrocephalus.
XII (1518).	<i>No evidence of hallucinations.</i>	No appropriate lesions or anomalies. Slight chronic internal hydrocephalus.
XIII (1540).	Auditory hallucinations surmised from some of patient's acts.	General mild atrophy and sclerosis of brain; no special emphasis in temporal regions, although the plication of the <i>right</i> temporal area was the simplest found in all cases examined in this series.
XIV (1571).	<i>No evidence of hallucinations</i> ; somatic delusions probably on a peripheral basis.	No appropriate lesions or anomalies. Cranial asymmetry, possibly acquired, and possibly related to somatic delusions.
XV (1574).	<i>No evidence of hallucinations.</i>	No appropriate lesions or anomalies.
XVI (1575).	No evidence of true hallucinations; remarkable pseudohallucinations ("overimagination").	No temporal-lobe lesions or anomalies. General cortical atrophy, but most marked behind Rolando and above Sylvius.
XVII (1583).	Visual, auditory and tactile hallucinations.	Left-hemisphere gyri much more simple in general architecture, especially parietal and at junction of occipital and temporal regions.
XVIII (1593).	<i>No evidence of hallucinations.</i>	General atrophy or hypoplasia of cortex, with emphasis in frontal and central regions.

NO.	SYMPTOMS.	ANATOMICAL CORRELATIONS.
XIX (1602).	Auditory hallucinations (or ideas of reference?) at first; later, paranoid picture.	Chronic internal hydrocephalus; chronic leptomeningitis of vertex; no correlations with auditory hallucinations, if such existed.
XX (1603).	<i>No evidence of hallucinations.</i>	Anomaly of superior temporal gyrus, like that of Cases II and III ( <i>clinical correlation absent</i> ).
XXI (1615).	Auditory hallucinosis (perhaps elaborate <i>Ge-dankenlautwerden</i> ).	Opercular atrophy, left.
XXII (1616).	Auditory hallucinations? (God's commands).	Superior temporal anomaly.
XXIII (1622).	Auditory hallucinations fairly constant.	No gross temporal-lobe correlation (the microscope shows considerable change).
XXIV (1625).	<i>No evidence of hallucinations.</i>	Temporal lobes simple in architecture, but no evidence of atrophy.
XXV (1634).	<i>No evidence of hallucinations</i> (visual?).	Grossly, negative.

There are 12 cases in the 25 which are recorded to have had hallucinations, although in a few instances it is possible that ideas of reference or other mental states have been mistaken for hallucinations. If we take the usual clinical ground that there are more hallucinations than the examiner is ever likely to prove, it is perhaps safer at first to count these all as hallucinations; our anatomical correlations could only show a lowered index if we adopt this course.

Of the 12, there are three cases which yield no convincing gross anatomical correlation.

Case VI is described as having had one episode of auditory hallucinations. This patient died in his 17th year (he was very possibly a high-grade imbecile), and the episode in question occurred 16 months before death.

Case XIX was an almost purely delusional case, but apparently had one episode of hearing imaginary enemies talking. There was no evidence in the gross anatomy of the brain of a special location of lesions in the temporal lobe. (Microscopically there

were mild lesions in many areas, but not marked in the temporal areas; in fact, microscopically the most severe lesions were those of the two postcentral gyri, possibly related with the delusions of "dynamo shocks," "heavy feelings in limbs," etc.)

Case XXIII is perhaps the best exception to the rule of temporal-lobe correlations with auditory hallucinosis, since the two cases just mentioned had mere brief episodes of hallucinosis, whereas this case must be counted as having numerous periods of frank hallucinosis through a period of 10 years. I am here forced to fall back on the fact that microscopic examination yielded considerable evidence of cell loss in the superior temporal gyri (although less than elsewhere, and especially less than in the parietal regions—the case was in the main a catatonic one).

Accordingly I conclude that the great majority of hallucinating cases of dementia præcox exhibit at autopsy and upon careful gross brain analysis rather striking evidences of temporal-lobe involvement. Exceptions are cases with single brief episodes of hallucinosis. The one apparently genuine exception (a paranoid and catatonic case, given to characteristic long periods of auditory hallucinosis) did show moderately severe microscopic lesions; but for unknown reasons the configuration or size of the temporal lobe had not been modified.

But, even if the validity of this correlation be granted, it may well be asked whether there are not numerous other cases which show marked temporal-lobe changes *without* auditory hallucinosis. On *à priori* grounds one must naturally answer this question affirmatively, and for a much wider field of cases than the dementia præcox group. Surely the conditions which favor the hallucinating process must be quite special, and it is highly improbable that any random injury to the temporal lobe, whether wrought in or after the period of development, should in and of itself start up auditory hallucinations. I therefore expected to find fairly numerous examples of temporal-lobe change without auditory hallucinosis, all the more because the histories and records of our patients are not always held to the highest level of accuracy.

The cases that illustrate non-correlation of gross anatomical changes found with hallucinosis number at most four:

Case IV, a hyperphantasia case, might well have shown auditory hallucinations, as well as what he did show, viz., delusions

and catatonic phenomena. Some of the hyperphantasia expressed by this patient was of a sort possibly to be ascribed on theoretical grounds to temporal-lobe involvement or to a simultaneous involvement of both parietal and temporal lobes.

Case X showed temporal lobes of unusually simple architecture, and they appeared to be subject to a moderate degree of atrophy. The only suggestive correlation so far obtained is mannerisms of speech, which I suppose it is conceivable to attribute in certain instances to some disorder of word or phrase storage. But the history of this case has unfortunately not yet been properly followed out.

Case XX, another hyperphantasia case, was one of a special group showing anomalies of the superior temporal gyri. There are indications in the history that this patient may have had auditory hallucinations, although I have left them out in the above table.

Case XXIV showed temporal lobes of unusually simple architecture, but no gross atrophy had been developed.

To sum up concerning the lack of expected hallucinosis: Four of 13 cases *without* auditory hallucinosis showed temporal-lobe changes, (1) a case with unduly simple temporal lobes without atrophy, (2) a case similar to (1) but with a moderate atrophy, (3) a case with general hypoplasia (and atrophy?) in which infra-Sylvian tissues especially shared, and (4) a case with superior temporal anomaly similar to others in the series (all the others showed hallucinosis, and this case may have). The latter two cases, mentioned in the previous sentence (*viz.*, Cases IV and XX), were cases of hyperphantasia, in which it is possible that temporal-lobe functions may play a part.

On the whole, the small percentage (three or four in 13) of cases having temporal-lobe changes without hallucinosis is, I think, smaller than one would *à priori* predict. One may argue therefrom that in dementia præcox patients temporal-lobe changes surprisingly often take such a form as to permit auditory hallucinosis.

Harking back to the correlation just preceding, it is clear that one may reason even more safely from the fact of hallucinosis to the probability of temporal-lobe disorder than from the existence of temporal-lobe disorder to the history of hallucinosis.

The point probably lodges in the fact that quite special conditions after all determine the occurrence of hallucinations, and that not every temporal-lobe disorder will serve. One must remember also that we are dealing with dementia præcox, and with dementia præcox only, in which disease possibly special stratigraphical disease occurs in which special cells alone are altered or destroyed.

With the two small groups of exceptions just mentioned, the general correlation of auditory hallucinosis with temporal-lobe disorder may be considered to be provisionally established, at least for the dementia præcox group.

#### CORRELATIONS OF PARIETAL-LOBE LESIONS WITH CATATONIA.

Following is a table designed to bring out the evidence for or against my formerly suggested correlation between catatonia and post-Rolandic (postcentral and other parietal) lesions. I have included all cases both catatonic and not catatonic; and in the right-hand column I have placed all evidences of parietal involvement, whether the cases were or were not catatonic.

TABLE XIV.

#### GROSS ANATOMICAL CORRELATIONS WITH CATATONIA.

NO.	SYMPTOMS.	ANATOMICAL CORRELATIONS.
I (1297).	<i>No certain evidence of catatonia</i> (outbursts of profane or obscene speech, quarrelsomeness, incoherent letters).	Left hemisphere above Sylvius and forwards of parieto-occipital fissure atrophic, but maximal atrophy is not parietal; post-Rolandic tissues of right side more atrophic than left.
II (1317).	Catatonia late in course (resistivism, mutism, refusal to eat, violence; later, apathy, manneristic speech, grimaces).	The only gross lesion behind Rolando was a tendency to occipital microgyria; <i>parietal correlations absent</i> .
III (1319).	Catatonia? (resistive but fearful, tube-fed at times).	No special parietal-lobe change (unless affected by the chronic internal hydrocephalus, more marked on left).



NO.	SYMPTOMS.	ANATOMICAL CORRELATIONS.
IV (1335).	Catatonia gradually sup- planted paranoic symptoms (catatonic excitements, manner- isms, fixed positions, inability to walk a straight line).	Parietal emphasis of the gen- eral brain atrophy (possibly hypoplastic at onset).
V (1383).	Catatonia (mannerisms, spasmodic movements of jaw, lid-closure).	Postcentral atrophy, more marked on right. Internal hydrocephalus, especially of mid portions of ventricles.
VI (1413).	Episode probably cata- tonic.	Callosomarginal anomalous folding above splenium on both sides.
VII (1472).	Nothing catatonic until development of pe- culiar stepping move- ments toward close of long course.	General slight brain atrophy.
VIII (1487).	Catatonia (three weeks' course).	No sclerosis or atrophy any- where; asymmetry of brain.
IX (1491).	Catatonia (head con- stantly uplifted in bed, mutism, <i>cerea flexibil- itas</i> , resistivism, nega- tivism).	Chronic internal hydroceph- alus; mild atrophy (aplasia?) of paracentral lobules.
X (1507).	Mannerisms of speech.	No parietal-lobe lesions.
XI (1509).	Catatonia (excitements, impulsive acts, mu- tism, resistivism, re- fusal of food, man- nerisms, limp mus- cles).	Moderate chronic internal hy- drocephalus, moderate parie- tal atrophy.
XII (1518).	Catatonia (periods of excitement, negativ- ism, resistivism).	Callosomarginal acquired le- sion, right; developmental disturbance of same region, left; chronic internal hydro- cephalus, more marked posteriorly.
XIII (1540).	Catatonic resistivism, mannerisms of pos- ture, impulsive vio- lence.	General mild atrophy and scler- osis of brain; maximal atro- phy (or aplasia?) of left angular gyrus; chronic in- ternal hydrocephalus, more marked posteriorly.

NO.	SYMPTOMS.	ANATOMICAL CORRELATIONS.
XIV (1571).	No evidence of catatonia (somatic delusions).	No parietal-lobe change.
XV (1574).	No evidence of catatonia.	No parietal-lobe changes.
XVI (1575).	Some symptoms are allied to catatonia (peculiar "tranquil" violent kicking of patients, etc.).	General brain atrophy (especially superior and inferior parietal regions).
XVII (1583).	Catatonia ( <i>cerea flexibilitas</i> , impulsive violence, resistivism).	Left-hemisphere gyri much more simple in general architecture than right, especially parietal and at junction of occipital and temporal gyri; moderate general brain atrophy; asymmetry.
XVIII (1593).	Catatonia (catalepsy, <i>cerea flexibilitas</i> , neologisms, mutism).	Postcentral atrophy, well marked, along with precentral and superior frontal atrophy.
XIX (1602).	No evidence of catatonia.	Marked chronic internal hydrocephalus; chronic leptomeningitis of vertex.
XX (1603).	No evidence of catatonia.	Marked chronic internal hydrocephalus; no special parietal involvement.
XXI (1615).	No evidence of catatonia (one "hysterical" attack).	Inferior parietal atrophy.
XXII (1616).	Catatonia (mannerisms, violence).	Chronic internal hydrocephalus (no special parietal involvement).
XXIII (1622).	Catatonia late in course (mutism, resistivism).	Superior parietal atrophy; left supramarginal atrophy most marked.
XXIV (1625).	Catatonia (resistivism, negativism, verbigeration, fixed attitudes, <i>cerea flexibilitas</i> ).	Chronic internal hydrocephalus confined to posterior halves of bodies of lateral ventricles; thinning of posterior part of corpus callosum; atrophy of inner surfaces of occipital lobes.
XXV (1634).	Catatonia ( <i>cerea flexibilitas</i> , mannerisms, impulsive movements).	Grossly, negative.

My clinical analysis yields 14 cases in which there seems to be no doubt that catatonic symptoms definitely appeared, and there are seven others in which catatoniform or allied symptoms are recorded. Four were quite negative.

Of the 14 definitely catatonic cases (and here I do *not* mean cases which would necessarily be placed in Kraepelin's catatonic *form* of dementia præcox, either in his formulation of 1899 or in that of 1913; but I refer to cases having *symptoms* of this general nature), 10 at once yielded parietal or other correlations behind the fissure of Rolando. Two of four others were the two grossly negative cases of brief duration (VIII and XXV) in which no gross *acquired* lesions of an atrophic nature could have been expected (*they did show microscopic changes*, as elsewhere mentioned).

This analysis leaves outstanding two cases with absence of gross correlations. II developed catatonia late, and the only gross lesion or anomaly behind Rolando was a tendency to microgyria of the occipital regions. XXII showed mannerisms and violence supposed to be catatonia; and the brain showed chronic internal hydrocephalus, but without emphasis upon any region behind Rolando.

If we count II and XXII as without correlation (although this is hardly a fair account of either), still the percentage of incidence of posterior-lying lesions in catatonia stands at 85 per cent.

Of the seven cases in which our interpretation must remain doubtful as to whether the symptoms were catatonic or not, but which may well have been catatonic, I find five with various posterior-lying lesions, and two without such correlation.

In four cases in which we can apparently exclude catatonic symptoms entirely, there were no posterior-lying lesions.

Analyzing from the standpoint of catatonia, accordingly, the parallelism is very striking between catatonia and lesions of the cortical arrival-platforms and the posterior association center. But, as in the case of hallucinosis and the temporal region, it is only fair to correlate in the other direction, from lesion to symptom. I will not here repeat what was said above under hallucinosis, to the effect that correlations are likely to be less strong when running from lesion to symptom than from symptom to lesion.

We seem to be able to exclude all gross lesions and anomalies of whatever kind (so far as posterior-lying lesions go) in Cases VIII, X, XIV, XV, XXV, of which the first and last (VIII and XXV) are the grossly negative cases in which we must rely on microscopic changes, and of which X showed merely mannerisms of speech, and XIV and XV showed no catatonia.

Thus there were 20 cases in which the brain showed either a special emphasis of posterior-lying lesions in the gross or involvement of these areas in a general or universal brain change.

Taking these in order we find all clearly catatonic except the following:

- I. (Outbursts of profane or obscene speech, quarrelsomeness, incoherent letters.)
- III. (Catatonia?—resistive but fearful, tube-fed at times.)
- VI. (Episode probably catatonic.)
- XVI. (Symptoms allied to catatonia.)
- XIX. (No evidence of catatonia.)
- XX. (No evidence of catatonia.)
- XXI. (One "hysterical" attack.)

Thus 65 per cent of the cases showing either general or special involvement of the posterior-lying areas were catatonic, and some suspicion attaches to 25 per cent more, leaving 10 per cent with absolutely negative correlations. These two cases, XIX and XX, were cases of marked internal hydrocephalus, but without special emphasis.

If these figures can be taken on their face-value, it may well be supposed that not every case which exhibits posterior involvement will necessarily produce catatonic symptoms to correspond and that very possibly special conditions (cytological, strati-graphical, microphysical) have to do with the appearance or non-appearance of these symptoms. In particular, however often catatonia is correlated with internal hydrocephalus, it does not yet appear that the conditions producing the hydrocephalus necessarily produce catatonia, although for unknown reasons they seem more likely to produce catatonia (as well as hallucinosis) than to produce delusions.

Special attention should be given to *cerea flexibilitas* as a catatonic symptom *par excellence*. Following are cases:

- IX. (Internal hydrocephalus; slight atrophy (aplasia?) of paracentral lobules.)
- XVII. (Moderate general brain atrophy, with apparently greater involvement of parietal region and zone of junction of temporal and occipital regions on left side (due to aplasia?).)
- XVIII. (Postcentral atrophy, well marked.)
- XXIV. (Internal hydrocephalus limited to posterior halves of bodies of lateral ventricles; thinning of posterior part of corpus callosum; atrophy of inner surfaces of occipital lobes.)
- XXV. (Grossly, negative; microscopically, positive case.)

In this series of cases with *cerea flexibilitas*, the tendency to parietal involvement is especially plain, and inasmuch as two of the four cases with lesions showed the gross emphasis of these lesions in or near the sensory arrival-platforms for touch and muscle-sense, it may well be supposed that certain conditions in those areas underlie *cerea flexibilitas*. From the nature of that symptom itself, it may be surmised that its relation to disordered kinaesthesia may some day be established.

#### CORRELATIONS OF FRONTAL-LOBE LESIONS WITH DELUSIONS.

Following is a table designed to bring out the truth or falsity of the *paranoia-frontal-lobe* correlation for which I endeavored to lay a foundation in the 1910 paper (see conclusions 12 and 14 from that paper, quoted above).

It should be said that I use the term *paranoia* in the sense of a symptom, *not* in the sense of an entity. Moreover, I use it in a broad sense to indicate "insane delusions" of all sorts, not in the narrower sense of "delusions of persecution." The correlation we here investigate is accordingly between the delusional character and brain conditions, between the acquired or dispositional tendency to false beliefs (or better, the habit of falsely believing) and disease process in whatever we may discover to be the believing-mechanism in the brain.

In parentheses in the left-hand column are placed also the data concerning hallucinosis, transferred in brief from the previous table.



TABLE XV.

## GROSS ANATOMICAL CORRELATIONS WITH PARANOIA (INSANE DELUSIONS).

NO.	SYMPTOMS.	ANATOMICAL CORRELATIONS.
I (1297).	Delusions, allopsychic (with hallucinosis).	Entire <i>left</i> hemisphere above Sylvius and forwards of parieto-occipital fissure atrophic; maximal atrophy about right first frontal sulcus; corpus callosum <i>not</i> thinned.
II (1317).	Delusions, religious, and fear, probably both autopsychic and allopsychic (with hallucinosis).	Left superior frontal gyrus smaller than right (developmental disorder with super-added atrophy?).
III (1319).	Delusions, religious (regarded as based on hallucinosis).	No special frontal-lobe correlate (unless affected by the chronic internal hydrocephalus, more marked on left).
IV (1335).	Delusions, allopsychic, fantastic, supernatural agency (without hallucinosis).	General atrophy of brain (possibly hypoplastic at outset), but with parietal and temporal emphasis.
V (1383).	No evidence of delusions (long-standing case with little early history).	General brain atrophy, more marked on right, and more marked forwards of Rolando. Internal hydrocephalus, especially mid portion.
VI (1413).	Delusions, allopsychic, perhaps dominantly autopsychic; change of character, possibly high-grade imbecile (one episode of hallucinosis).	Tendency to frontal atrophy, more marked on left.
VII (1472).	Delusions, allopsychic, persecutory, with tendency to hyperphantasia (with questionable hallucinosis).	General hypoplasia, or more probably atrophy, of brain; second and third frontal gyri tend to show greater atrophy.
VIII (1487).	Delusions, religious, self-accusatory, at first; later characteristically catatonic.	No gross lesions; asymmetry of brain (three weeks' duration).

NO.	SYMPTOMS.	ANATOMICAL CORRELATIONS.
IX (1491).	History inadequate; a few delusions probable, allopsychic (no history of hallucinosis).	No frontal-lobe lesions, except tendency to mesial thinning (but this was more marked about paracentral lobule); chronic internal hydrocephalus.
X (1507).	Delusions, allopsychic.	Frontal atrophy or aplasia of milder degree than the temporal-lobe atrophy or aplasia (which was itself moderate).
XI (1509).	Delusions may possibly be inferred from hyper-religiosity (hallucinosi inferred from attitudes).	Moderate frontal atrophy (along with central and parietal atrophy); moderate chronic internal hydrocephalus.
XII (1518).	Delusions—allopsychic?—inferred from certain replies (without hallucinosis).	Left-sided frontal aplasia; slight chronic internal hydrocephalus.
XIII (1540).	Delusions, sexual, as well as others inferred from evasive and reticent attitude (hallucinosi surmised).	Mild atrophy (or aplasia?) of both superior frontal regions (mesial as well as lateral).
XIV (1571).	Fantastic somatic delusions, probably on a peripheral basis (without hallucinosis).	No special frontal involvement; but marked asymmetry (distortion?) of brain.
XV (1574).	Delusions, autopsychic, often fantastic in content (without hallucinosis).	Frontal (as well as parietal) opercular atrophy.
XVI (1575).	Fantastic delusions, allopsychic—and autopsychic? (without hallucinosis).	General cortical atrophy, especially superior and inferior parietal.
XVII (1583).	Delusions, allopsychic, somatic (with hallucinosis).	No special frontal-lobe involvement (except that left is simpler than right; this disparity is still greater in parietal and at junction of occipital and temporal regions).

NO.	SYMPTOMS.	ANATOMICAL CORRELATIONS.
XVIII (1593).	Delusions, sexual, later grandiose (without hallucinosis).	General cortical atrophy, especially superior frontal and central (corpus callosum not thinned).
XIX (1602).	Fantastic delusions, allopsychic, autopsychic, somatic (hallucinosis questionable).	No special frontal involvement (except for marked chronic internal hydrocephalus); thinning of posterior part of corpus callosum.
XX (1603).	Delusions, allopsychic, perhaps dominantly autopsychic; hyperphantasia (without hallucinosis).	No special frontal involvement (except for marked chronic internal hydrocephalus); marked chronic leptomeningitis over left frontal operculum.
XXI (1615).	Delusions—ideas of reference; perhaps elaborate <i>Gedankenlautwerden</i> (question of hallucinosis).	No frontal-lobe involvement, except opercular atrophy, more marked on left; inferior parietal atrophy.
XXII (1616).	Delusions, autopsychic, allopsychic? (hallucinosis doubtful).	No frontal-lobe involvement (except possible effects of chronic internal hydrocephalus).
XXIII (1622).	Delusions, autopsychic, allopsychic (with hallucinosis).	No frontal correlation.
XXIV (1625).	Delusions questionable—ideas of reference (without hallucinosis).	Inferior frontal gyri aplastic? or atrophic?
XXV (1634).	Delusions, allopsychic (without hallucinosis).	Grossly, negative.

There is an *embarras de richesse* in the business of anatomical correlations with delusion-formation. Both delusion-formation and frontal-lobe atrophy are common phenomena, and perhaps we hesitate to admit their correlation (to the end of establishing any sort of causal relation) just by reason of the frequency of their appearance.

There is but one case in the series in which there is no evidence of delusion-formation; that case (V) exhibited a generalized brain atrophy, more marked on the right side and *forwards* of the

fissure of Rolando, as well as internal hydrocephalus, especially of the mid portions of the lateral ventricles. The history, duration of disease, and the very age of the patient are all unknown to us, although (as the case-description shows) there can be little question of the appropriateness of the diagnosis dementia præcox from the terminal symptom picture. I should hesitate to break the force of any correlations concerning delusion-formation on the evidence of this case. Note is made of a certain circumstantial loquacity occasionally breaking forth and of various peculiar ways of thinking, which may represent residuals of a frontal-lobe disorder early more marked.

Of the 24 cases with delusion-formation more or less in evidence, seven exhibited no gross frontal-lobe disorder. Two of these are the two entirely negative cases (from the gross point of view), viz., VIII and XXV. Both these cases, as elsewhere mentioned, yielded ample microscopic evidence of frontal-lobe disease.

The five remaining cases of delusion-formation without frontal-lobe correlations deserve special comment.

XIV was a case presenting well-marked fantastic delusions of a somatic nature (which I consider probably had a peripheral basis). Like several others of the "hyperphantastic" group, this case exhibited no special frontal involvement, but, unlike several, failed to show special parietal involvement (see separate discussion of the hyperphantasia group, as also remarks in the case-description above). I am inclined to believe, with my colleague, Dr. H. M. Adler, that the future may show that the peculiar delusions of bodily distortion may be somehow correlated with acquired or progressive skull-distortion. Such is the peripheral basis which I mentioned above; but when the skull itself is the seat of changes, it is a question of physics which parts of the brain will respond symptomatically. I prefer to align this case with several reported in my communication "On the Somatic Sources of Somatic Delusions."

XVII was another case of somewhat less fantastic delusions of a similar somatic nature. The only delusions of an apparently somatic nature were some of poisoning, which may well have been founded on hallucinosis ("poison in saliva"). As I read the case, possibly all the delusions were founded on hallucinosis, or

actual morbid somatic phenomena, or on a fanciful rendering of normal somatic phenomena. Anatomically, the left frontal region was of simpler construction than the right, but there was no evidence of frontal-lobe atrophy.

XXI was also a case of unusual nature, and the attitude was possibly largely one of elaborate *Gedankenlautwerden* (long archaic prayers in French, etc.) in which there may have been a strong hallucinational nucleus. There was in this case no special frontal involvement, except that the left inferior frontal region was atrophic.

XXII yielded autopsychic and possibly allopsychic delusions, but showed no special frontal-lobe disorder, unless we may suppose effects wrought by internal hydrocephalus. The delusions mentioned are somewhat doubtful, and are perhaps in part due to auditory hallucinosis ("heard God's commands"); and the case early turned into a strongly catatonic case.

XXIII was a case of pronounced autopsychic delusions, combined with (or largely based upon?) somatic delusions. The hallucinational and catatonic symptoms were equally marked. Anatomically, the case falls more in the parietal group. Microscopically, there was considerable evidence of frontal involvement, but again the emphasis was parietal.

No one of these cases is strong enough to break the general correlation; indeed, three of the cases of apparent exception to the frontal-delusional correlation, viz., XIV, XVII, and XXI, go far to illustrate the concept of what might be termed sensorial delusions, considered elsewhere (hyperphantasia group).

#### CASES WITH FANTASTIC DELUSIONS (HYPERPHANTASIA).

I was led to distinguish, above, the large group of cases of ordinary delusion-formation from a small group in which a varied succession of absurd and fantastic delusions was developed. Upon theoretical grounds, holding to the correlation of ordinary delusion-formation with lesions of the anterior association-center, I was inclined to consider that the basis of the hyperphantasia might lie in the posterior association-center. Let us now consider this question on its merits.



## HYPERPHANTASIA.

- IV. Lesions (aplasia and atrophy) universal, but more marked in occipital and temporal regions.
- VII. Lesions (aplasia and atrophy) universal, but more marked in temporal and middle frontal regions.
- XVI. Lesions (atrophy) universal, but more marked in regions behind Rolando and above Sylvius, and most marked in superior and inferior parietal regions.
- XX. Internal hydrocephalus and left-sided superior temporal anomaly.
- XXI. Internal hydrocephalus (doubtful) and parietal atrophy.

In the above five cases the delusions were such as to attribute fantastic features to the environment. In two others the fantastic features were attributed to various parts of the bodies of the patients.

XIV. Correlation proposed with (acquired?) asymmetry of skull.

XIX. Posterior-column degeneration of spinal cord; internal hydrocephalus; chronic leptomeningitis.

In the entire group of seven cases it will be observed that universal lesions or conditions of unusual character prevail, as a rule either generalized atrophy or internal hydrocephalus (though not both). Accordingly, no area can be safely excluded from a share in the effects. Hydrocephalus in any event seems to affect the posterior-lying portions of the brain more than the anterior-lying portions. Where gross atrophy appears, it is exhibited as a rule most markedly in the parietotemporal regions.

Concerning the somatic hyperphantasia cases, one should not dogmatize, although I seem to find adequate somatic or sensorial nuclei for the delusions developed by these cases.

For the non-somatic group of hyperphantasia cases, I am inclined to vindicate the parietotemporal correlation, not omitting to concede, however, that the frontal regions are also as a rule to some extent involved.

## VI. CONCLUSIONS.

1. The writer has followed up his earlier work on the dementia præcox group (1910) with a more systematic anatomoclinical study of 25 cases, having a view to (a) definite conclusions as to the structurality ("organic nature") of the disease, and (b) correlation of certain major symptom groups (delusions, catatonic symptom groups, auditory hallucinosis) with disease of particular brain regions.

2. As to (a), the *structurality of dementia præcox*, the writer feels that the disease must be conceded to be in some sense structural, since at least 90 per cent of all cases examined (50 cases, data of 1910 and 1914) give evidence of general or focal brain atrophy or aplasia when examined post mortem, even *without* the use of the microscope.

3. Moreover, *with* the use of the microscope, the problem of the normal-looking remainder can perhaps be solved, since the only two normal-looking brains<sup>7</sup> in the 1914 series of 25 yielded abundant appearances of cell-destruction and satellitosis in the cerebral cortex, which had not yet had time to be registered in the gross (cases of three weeks' and two months' duration respectively).

4. The method of anatomical analysis in the new series is a more systematic one than has been hitherto employed, involving careful gross description of the fresh brain; careful preservation (by suspension from basal vessels) in formaldehyde solution; systematic photography to scale of the superior, inferior (cerebellum removed), lateral, and mesial aspects before and after stripping the pia mater; study of all aspects of the brain as spread side by side in photographic form; further study of the preserved brains in the light of the photographic study; and eventual cytological or fiber studies of paired structures showing possible atrophy or aplasia.

5. The neuropathologist making such a brain analysis shortly discovers that there is often more to be learned from the gross than from the microscopic appearances, since, of two gyri, the one measurably smaller than the other (and therefore probably agenic, aplastic or atrophic), the microscopic appearances may often be hard to diagnosticate, as the normal-looking gyrus at the time of death may be just undergoing a satellitosis actually indicating more disease than its shrunken fellow.

6. Nevertheless, the gross analysis gives one perfectly convincing evidence of some kind of lesions, leaving to other methods of study the decision as to the congenital or acquired nature of these lesions. Some 14 of the 25 cases may be regarded as in some sense maldevelopmental, so as to arouse the suspicion that the acquired atrophy was grafted on top of a congenital agenesia or aplasia; but, in the opinion of the writer, aplasia is indicated rather than

agenesia: the potential victim of dementia præcox is probably born with the normal stock of brain cells, although their arrangement and development are at times early interfered with.

7. The atrophies and aplasias, when focal, show a tendency to occur in the left cerebral hemisphere. The coarse atrophy is usually of only moderate degree, and often does not appreciably alter the brain weight, at least outside the limits of expected variation. In fact, the heart, the liver, the kidneys, and the spleen tend to show greater loss in weight than does the brain.

8. More remarkable than the atrophy and aplasia of the cortex is the high proportion of cases of internal hydrocephalus (at least nine cases) uncovered by the systematic photographic study of frontal sections.

9. There is no evidence that this internal hydrocephalus is due to generalized brain atrophy. It is possible that it begins more posteriorly. It is probable that it does not mechanically so much affect the frontal lobes. It is associated with cases of long duration, although not with all cases of long duration, and was never found in cases of brief duration. Clinically, the hydrocephalic cases are uncommonly catatonic, and the cases of marked generalized hydrocephalus were as a rule victims of hallucinations. Delusions, except fantastic delusions, were not prominent in this group. The clinical courses of these hydrocephalic cases were more than usually active and mutable, and were often interrupted by remissions.

10. The hydrocephalic brains were not in other respects particularly open to the suspicion of congenital disease; and, without adequate proofs, the writer is inclined to consider the hydrocephalus to be often an acquired hydrocephalus.

11. An ardent supporter of congenital features might claim that 19 of the 25 brains showed some sort of maldevelopmental defect; one impartial witness thought that 14 showed such; and even if all nine cases of hydrocephalus be taken as acquired, we remain with 11 cases bearing pretty certain evidence of maldevelopmental defect. On the other hand, all but six cases showed signs of acquired lesion, and these six showed various microscopic changes of doubtful meaning, but certainly acquired.

12. One remains with the general impression that gross alterations are almost constant and microscopic changes absolutely con-

stant, and that the high proportion of gross appearances suggesting aplasia means that structural (visible or invisible) changes of a maldevelopmental nature lie at the bottom of the disease process. But this suspicion of underlying maldevelopment is only a suspicion, although a strong one, and the first factor for the theory of pathogenesis to explain is the gross and microscopic changes as they present themselves in the full-fledged case.

13. Aside from left-sidedness of lesions and internal hydrocephalus, very striking is the preference of these changes to occupy the association-centers of Flechsig. For this there is probably good *à priori* reason in the structure, late evolutionary development, and consequent relatively high lability of these regions. The interest of these findings is still greater in the functional connection (see below).

14. In concluding this summary of the anatomical side of the study, the writer cannot forbear adding that he supposes many neurologists, hearing of "lesions," will at once imagine extirpatory lesions of a Swiss-cheese appearance or areas like those of tuberous sclerosis. At the risk of being charged with *naïveté*, the writer would again here insist that the lesions described, though never beyond the range of a skilful anatomist, are of a mild atrophic nature or in the nature of aplasias, requiring care and deliberation in their description and explanation, and often hard to grasp except where photographs of all sides of the brain may be compared at once and reference then made to the brains themselves. These lesions do not effect global lacunæ in the cortical neuron systems, but they are of a more finely selective character. Under the microscope it may be difficult to say, without elaborate micrometry, that one area is worse off than another; but convincing evidence of the gross convolutional extent of the process is got by the naked eye and by the finger.

15. The writer regards this work as putting the burden of proof on those who claim the essential functionality of dementia præcox, and is at some pains to couch objections to one formulation of these changes as "incidental," and to another, as "agenesic." Nevertheless, the writer would not necessarily deny the value of those formulations which look on these cases as cases of faulty adaptation to environment.

16. As to (b), the *functional correlations* of this study, the results may be summed up by saying that strong correlations have been found to support the writer's former claims that (1) delusions are as a rule based on frontal disease, and (2) catatonic symptoms on parietal-lobe disease. An equally strong correlation (3) has now been found between auditory hallucinosis and temporal-lobe disease.

17. The writer's previous work had suggested a correlation between frontal-lobe disease and delusion-formation. This correlation is not so decided in the present series, since, although perhaps only one of the 25 cases failed to exhibit delusions, seven of the remaining 24 failed to show frontal-lobe lesions. However, two of these seven, though grossly negative, were microscopically positive enough.

18. The findings indicate, accordingly, that there is a group of delusional cases such that even long duration does not determine a frontal emphasis of lesions. Five cases represent this exceptional condition: three of these five are probably best interpreted as cases of hyperphantasia in which, both *à priori* and by observation, frontal lesions are not characteristic.

19. On the whole, the correlation between delusions and focal brain atrophy (or aplasia capped by atrophy?) is very strong, particularly if we distinguish (1) the more frequent form of delusions with frontal-lobe correlations from (2) a less frequent form with parietal-lobe correlations.

20. The non-frontal group of delusion-formations, the writer wishes to group provisionally under the term *hyperphantasia*, emphasizing the overimagination or perverted imagination of these cases, the frequent lack of any appropriate conduct-disorder in the patients harboring such delusions, and the *à priori* likelihood that these cases should turn out to have posterior-association-center disease rather than disease of the anterior association-center. This anatomical correlation is in fact the one observed.

21. The writer's previous work had suggested a possible correlation between catatonic phenomena and parietal (including post-central) disease: 10 of 14 definitely catatonic cases yielded parietal or other post-Rolandic lesions; two were grossly negative but microscopically altered; and indications of correlation appeared also in the remaining two. Five of seven clinically some-



what doubtfully catatonic cases yielded similar correlations. Four clinically non-catatonic cases yielded no parietal correlations. (It is worth while insisting that "catatonia" is here used to refer to a symptom, *not* to an entity or clinical group.)

22. Special interest attaches to *cerea flexibilitas* as a clearly definable form of catatonic symptom: four of five cases yielded gross parietal lesions. The fifth case was one of the entirely negative cases in the gross, but showed very marked postcentral satellitosis microscopically. Two of these cases showed the gross emphasis of lesions in the postcentral gyri, thereby hinting at an explanation of *cerea flexibilitas* along the lines of a reaction to altered kinæsthesia or an altered reaction to normal kinæsthesia (depending upon such true analysis of intragyral cortex-function as the future may bestow).

23. *A priori* one might expect a correlation between the characteristic auditory hallucinosis found in many cases of dementia præcox and temporal-lobe lesions. In point of fact, nine of 12 hallucinated cases yielded temporal-lobe atrophy or aplasia; and actually only one of the three others is a good exception to the rule (from the clinical standpoint), to say nothing of the fact that this case had ample microscopic changes in the temporal lobe.

24. Of the 13 *non-hallucinating* (auditory) cases, only three, or at most four, could be said to have temporal-lobe lesions suggesting the possibility of hallucinosis; here we may appeal to the inadequacy of clinical work, or, better, to the non-suitability of the lesions, since no one would assert that we yet have any idea of the precise and intimate temporal-lobe conditions which permit hallucinations.

25. In these functional connections, the more recent formulations of Kraepelin and of Bleuler have been reviewed, although the entire work was done without the benefit of their analyses. The present formulation appears consistent enough with either. It would seem that Kraepelin regards a correlation between auditory hallucinosis and temporal-lobe disease as already highly probable from the literature. He also goes so far as to incriminate the "central" region for motor disorders. But the present suggestions as to the possible kinæsthetic relations of catatonia and the special (frontal and parietal) correlations with delusion-

formation are not suggested by Kraepelin from the literature available.

26. It is interesting to note that further study by the Munich workers seems to have drawn attention away from the *infrastellate* cortical changes sketched by Alzheimer for catatonia in 1897 to various *suprastellate* changes. The microscopic work done in the present study in connection with certain grossly negative cases indicates that the early phases of the process may very often look as if infrastellate change was to be the most striking product of the disease. This is perhaps due to a richer original supply of glia cells in these infrastellate layers. Later, when the process is less acute, it may often be found that suprastellate cell losses are much more in evidence than any striking infrastellate change.

27. As for the general position which this work would assume toward the functional conclusions of Bleuler, it would seem that a histopathological basis for "dissociations" or "schizophrenia" could be somewhat readily provided by the lesions found, since these are for long periods mild enough and sufficiently confined to the finer cortical apparatus to provide for the exquisite mental changes of most cases. The main neuronic systems are often permanently preserved, leaving an irregularly and slightly simplified cortical apparatus, in which a few cell changes would naturally throw out of coordination a great deal of still intact apparatus. But the whole process often remains so mild as to permit reestablishment of relatively normal functional relations on a slightly simplified basis, the whole to be disturbed once more on the occasion of the death or disease of a few more cells. Very striking is the fact that the cells not attacked are, so far as we can see, normal enough.

28. This work is rather a study of genesis than of etiology, in the sense of modern medical distinctions between these branches of inquiry. It is a modest inquiry into factors, and does not rise to the height of ascribing causes. The writer will refer merely to some paragraphs in the text as to a possible ontological position concerning structure and function which the future may take. The deplorable thing is that some structuralists throw out of court all functional data and some (rather more!) functionalists tend to underrate the possible contributions of anatomy to this field. Luckily, science nowadays cannot long proceed merely *à la mode*.

29. In particular, to sum up, I would call especial attention to the following points: (1) The constancy of mild general or focal atrophies in cases lasting long enough to yield these; (2) the tendency to an exhibition of lesions somewhat more markedly in the left hemisphere; (3) the preference of the lesions for the "association-centers" of Flechsig; (4) the high correlation of auditory hallucinosis and temporal-lobe lesions, as also (5) of catatonia and parietal lesions (*cereæ flexibilitas*, especially postcentral), and (6) of the more frequent form of delusions and frontal-lobe disease; (7) the possible existence of a hyperphantasia group with parietal correlations, and of (8) a large internal hydrocephalus group with catatonic and hallucinotic correlations rather than delusional. A few more points can be got from the description of the accompanying plates.

#### VII. DESCRIPTION OF PLATES.

A selection of photographs has been made from the complete collection to illustrate partially the points made in the text. The collection of which these photographs are examples contains now upwards of 7000, made systematically from over 500 brains representing cases of (a) mental disease (courtesy of various state institutions for the insane, especially Danvers and Boston), (b) epilepsy (courtesy of the Monson State Hospital), (c) feeble-mindedness (courtesy of Massachusetts School for the Feeble-minded), (d) criminality and other medico-legal cases (courtesy of the Suffolk District Medical Examiner), and (e) normal and miscellaneous nature (courtesy of various institutions and physicians).

The photographs have been made (1) with the pia mater *in situ*, (2) with the pia mater stripped, and (3) in frontal sections in chosen planes. The anatomical preparations have been made largely by Dr. Annie E. Taft, Custodian of the Neuropathological Collection, Harvard Medical School, serving as a special investigator under the State Board of Insanity under the direction of the writer as Pathologist to the Board.

The photography has been executed, save in a few instances, by a professional photographer, Mr. Herbert W. Taylor.

Considerable technical aid has been contributed by the Department of Neuropathology of the Harvard Medical School, largely from gifts by Miss Katherine E. Bullard and Mrs. Zoe F. Underhill. The contributions from the Underhill foundation have been made largely to secure evidence of normality of the brain in certain cases, as her gift was for the study of non-nervous factors in nervous and mental disease.

Plate I illustrates one of the most marked degrees of *generalized atrophy* found in the series. There is, however, a tendency to *focal emphasis of atrophy* in the inferior parietal region. As usual, the *left hemisphere* was more affected than the right. Death at 34 years, onset at 22, course of 12 years. Paranoid, later catatonic (Case XXIII).

Plates II and III present a good example of the striking *contrast* often shown *between the left and right hemispheres*, in which event the *left hemisphere* almost always presents *more atrophy* than the right. It would hardly be supposed that Plates II and III represented hemispheres from the same brain. This case was one in which disorder of glands of internal secretion may be safely asserted. Death after a course of nine months terminated by diphtheria (Case XVII).

Plates IV and V present mesial views from a brain (Case IV) which weighed 985 gm. at death (age 32, duration of symptoms 14 years, with a previous attack 20 years before death). Patient was an adequate shoe-operative, and there seems to have been no suspicion of feeble-mindedness in the sense of a congenital condition. Yet the heart (145 gm.), the aorta, the liver (945 gm.), as well as the spinal cord, were smaller than normal. Possibly there was a true agenesis with numerical loss of elements. Perhaps the case was rather one of aplasia (the writer's opinion). Case VII was a similar case (brain weight 955 gm., duration of symptoms 26 years, onset at 28), in a normal-school graduate teacher.

Plates VI to X are from Case II (death at 44, duration of symptoms 21 years).

Plates VI and VII are presented to show types of anatomical analyses. Attention is attracted to the small annectant-like gyrus in the left frontal lobe through which the line passes in Plate VI. Inspection shows that the left frontal lobe is narrower than the

right. Frontal section shows (Plate VII) that this idea is not deceptive, and that there is beyond question less substance to the left superior frontal division of the frontal region than to the right. Microscopic study (not here presented) would then be required to show whether this difference is only apparent and possibly due to the spreading over of superior frontal cortex type to a locus below the first frontal sulcus. This does not appear to be the case in the present examples. But, even if it were, the fact remains that the whole left frontal region is smaller than the right, at least in its forward part (see second frontal section, in which the difference has practically disappeared). Here, then, a contention is established that there is, anatomically at least, something to explain, be it agenesis, aplasia, or atrophy. Microscopically, there is much gliosis, though it is hard or impossible to tell whether more in sections from left or from right. But that there were fewer cells to start with in the left superior frontal gyrus than in the right can perhaps not be safely asserted; the question of agenesis *versus* aplasia is left unresolved both in the gross and in the microscopic examination, but may possibly be resolved by micrometric methods (size of cells on the two sides).

Plates VIII to X (from the same case as VI and VII) show another type of anomaly, of which there were five good examples in the whole series. The anomaly consists in an apparent "burial" of the left superior temporal gyrus. Frontal section (Plate X) shows these conditions in cross-section. The section is taken through a plane in which a local dimpling of the *right* superior temporal gyrus is also shown. (See below for another example of this anomaly.) By microscopic examination of the same loci in each superior temporal gyrus, it is possible to make a *rationale* as to which part of the gyrus has suffered most. Should one, however, rely on microscopic examination alone (without reference to the gross anomaly), the discovery of more or less well-marked gliosis in both gyri (the actual finding) would obscure the true issue as to initial agenesis or aplasia. Micrometric studies are required for this latter most important issue.

Plates XI to XIII are from Case III (death at 31, duration of dementia-præcox symptoms eight years), possibly a high-grade imbecile.



Plates XI and XII show a more pronounced example of the "burial" anomaly of the left superior temporal gyrus, together with a certain amount of cortical atrophy (aplasia?, agenesis??).

Plate XIII demonstrates the great difference between the left and right superior temporal gyri. There is also a certain degree of internal hydrocephalus, which it is to be noted is more marked on the side of the temporal anomaly. The left Sylvian fossa has much wider spaces than the right.

Plates XIV to XVI are from three different cases, and illustrate hydrocephalus.

Plate XIV was from Case XIX (possibly paraphrenia phantastica of Kraepelin's 1913 formulation), with onset at 34, duration of 28 years, death at 62. The brain was probably over weight at death (1315 gm., body length 157 cm.).

Plate XV was from Case XX, in which death was due to a massive cerebral hemorrhage. But (1) hydrocephalus and (2) superior temporal anomaly can still be made out. Death at 67, onset at 33 or younger.

Plate XVI was from Case XXII (onset at 26, death at 59). There was a small cerebral hemorrhage in this case also; possibly also a trace of the left-sided superior temporal anomaly shown in other cases.

Plates XVII to XIX are from one case (XII), with onset at 16 and death at 45. This case is being subjected to more intensive study, with the object of resolving the problem which the brain presents. Plate XVII gives the basal view, and shows what may be termed *cruciate asymmetry*. The *left* frontal region (at the right in the plate) is obviously smaller than the *right* (this is confirmed also by frontal section); but the *right hippocampal gyrus* (to the left in the plate) is obviously smaller than the *left*. Plates XVIII and XIX present mesial views of the same brain, in which the hippocampal disparity stands out well.

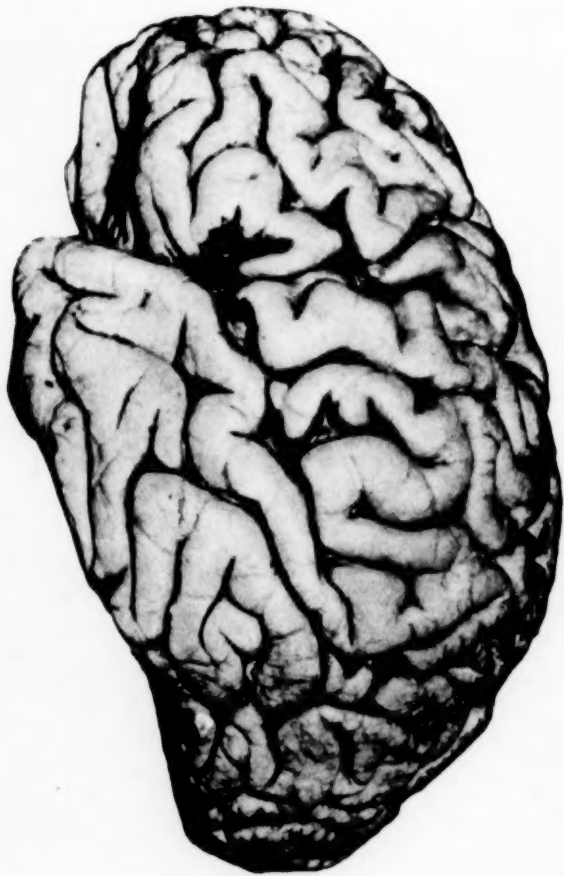
Can this be an example of agenesis or aplasia in which the disturbing factor acted prior to or during the decussating phase of the nervous system? That such may be the case is indicated by the reflection that most of the connections with the frontal region are very probably indirect or crossed connections, whereas the hippocampal connections are perhaps largely uncrossed connections.

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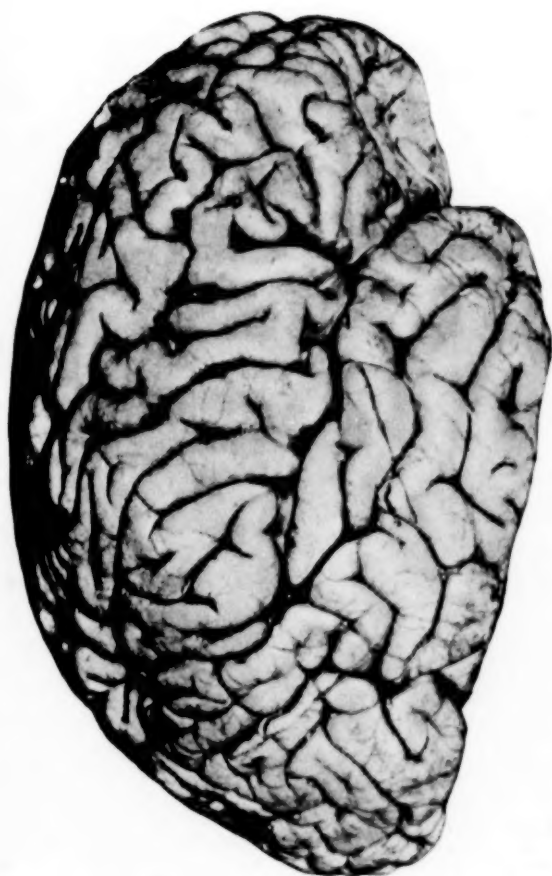
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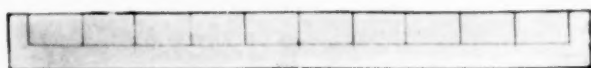
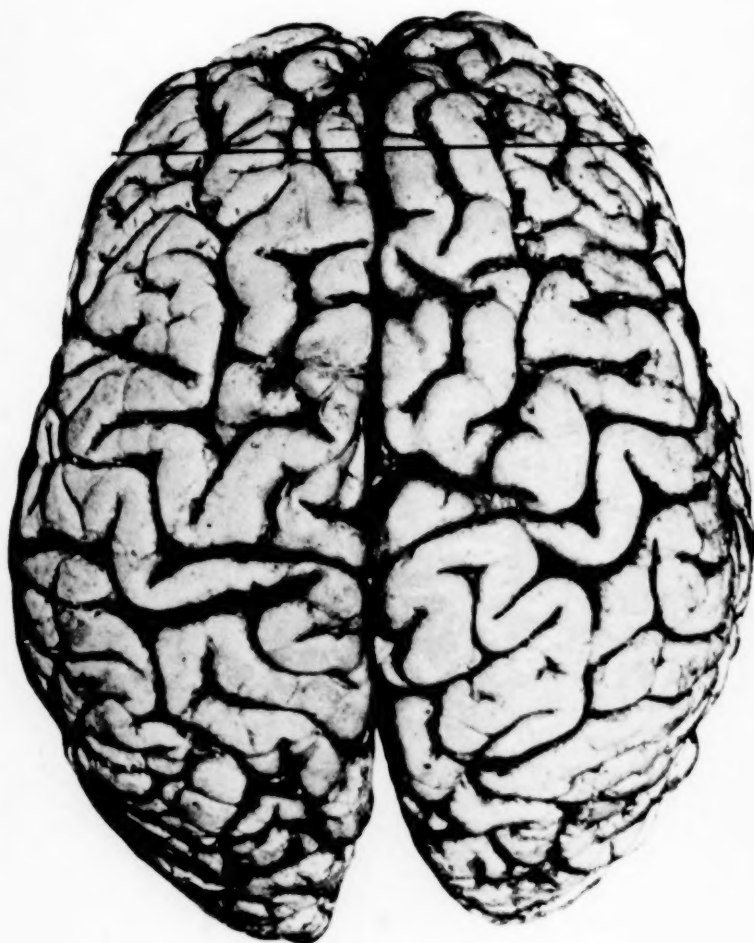


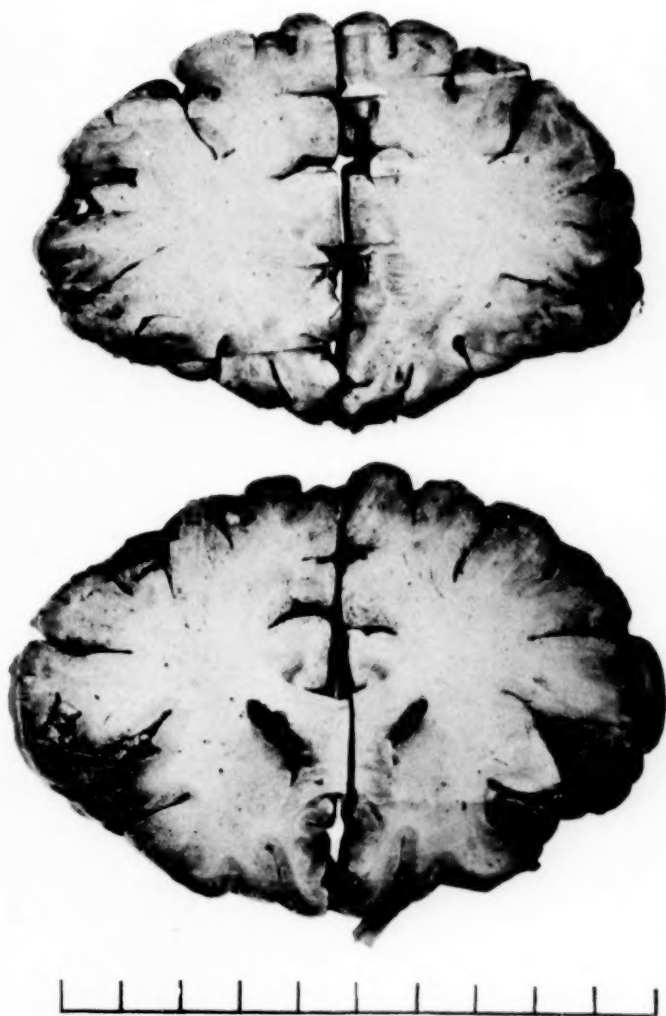




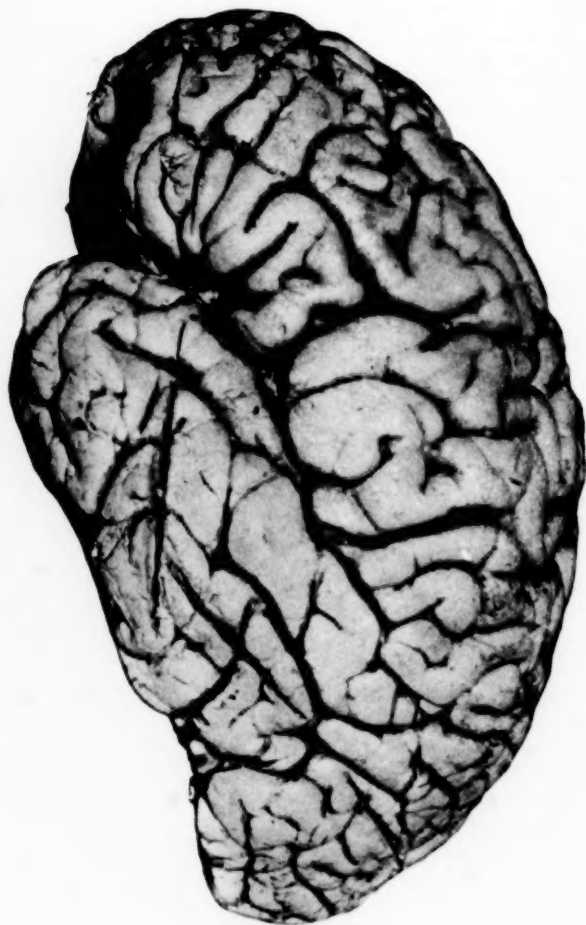


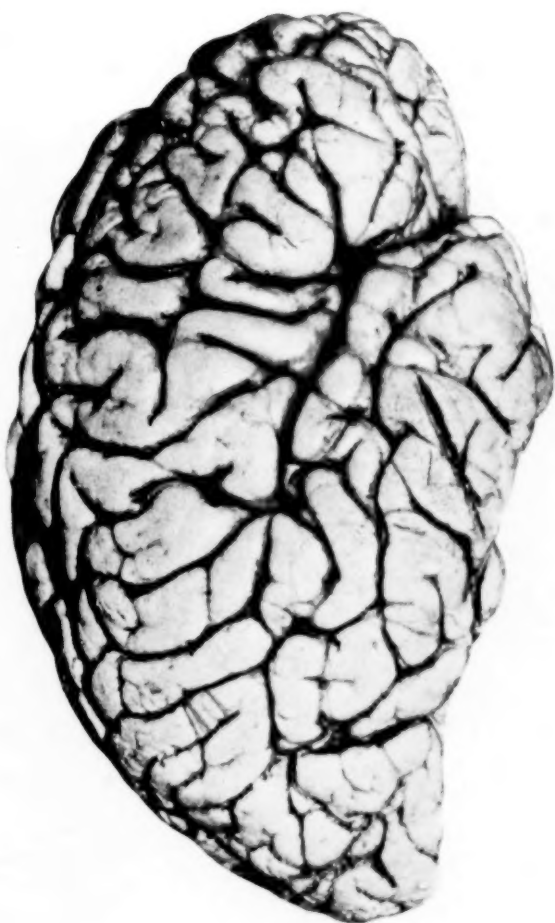




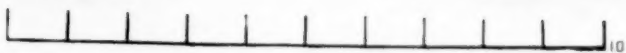






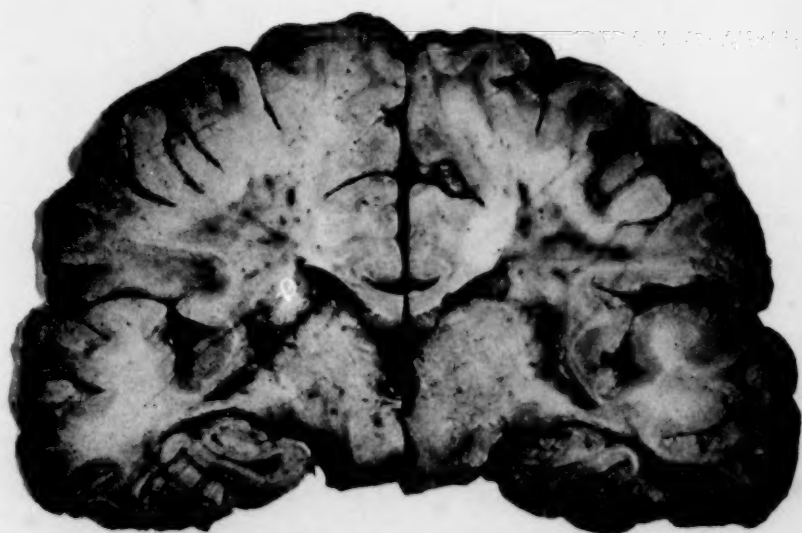


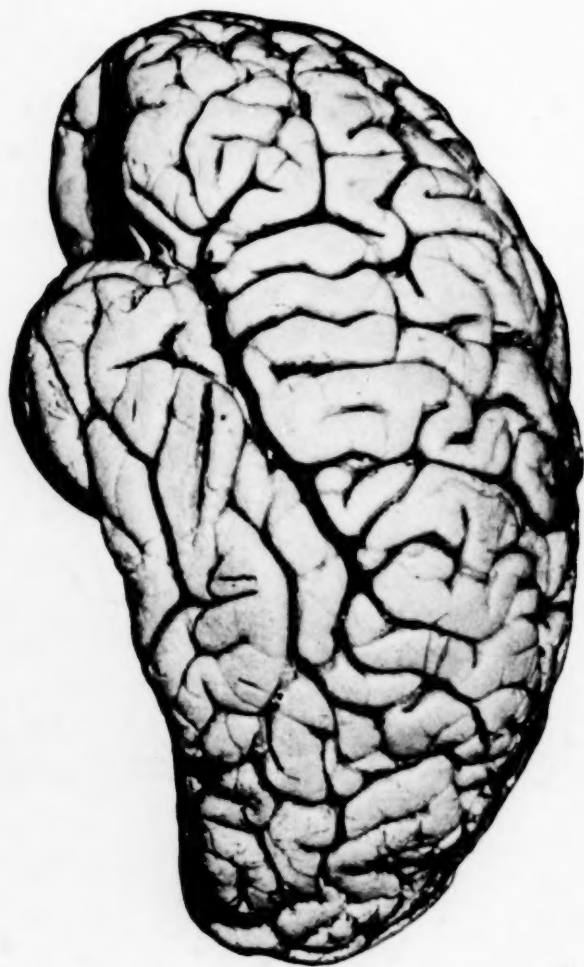
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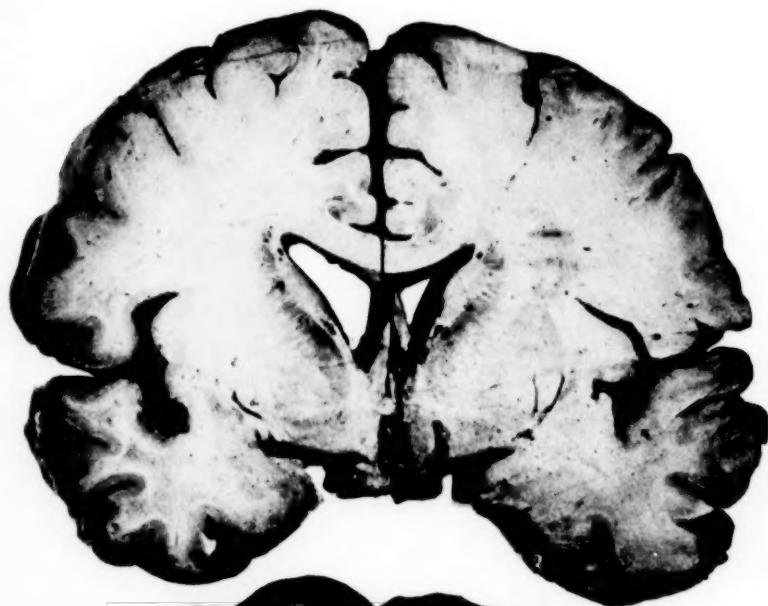
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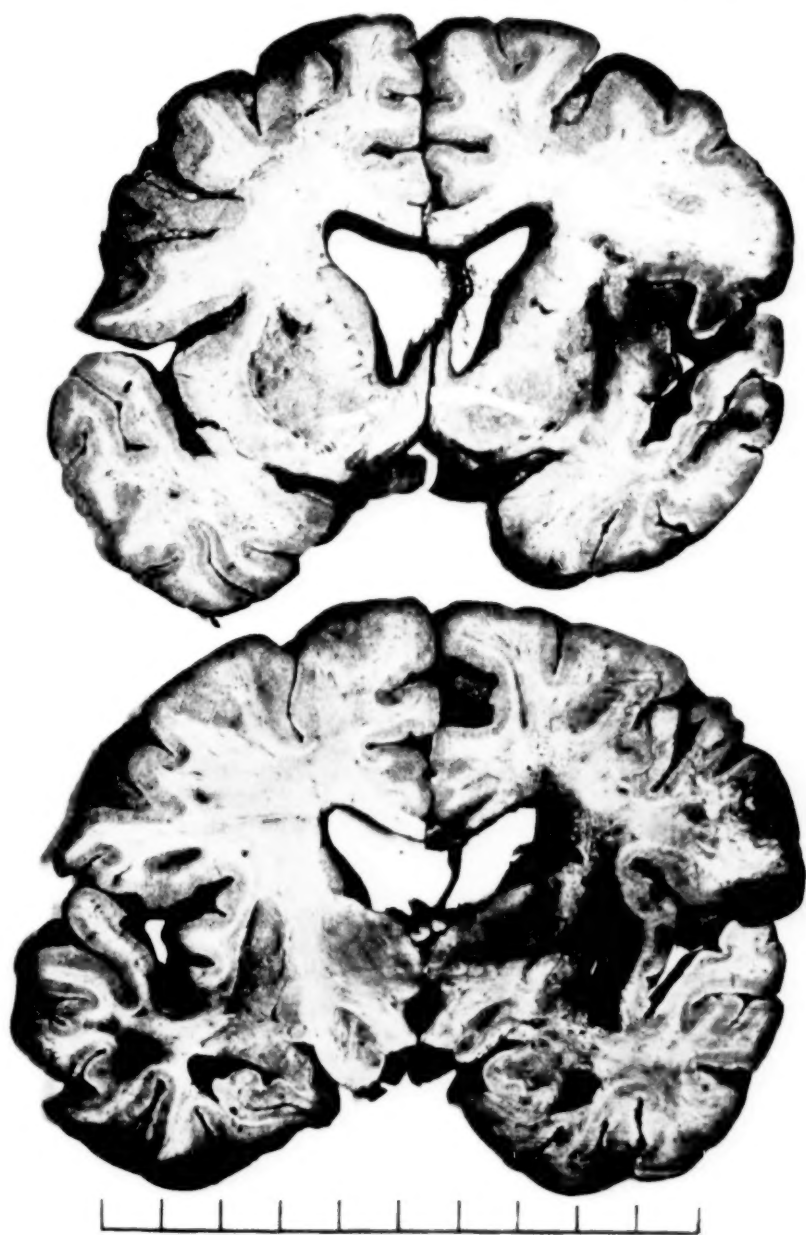






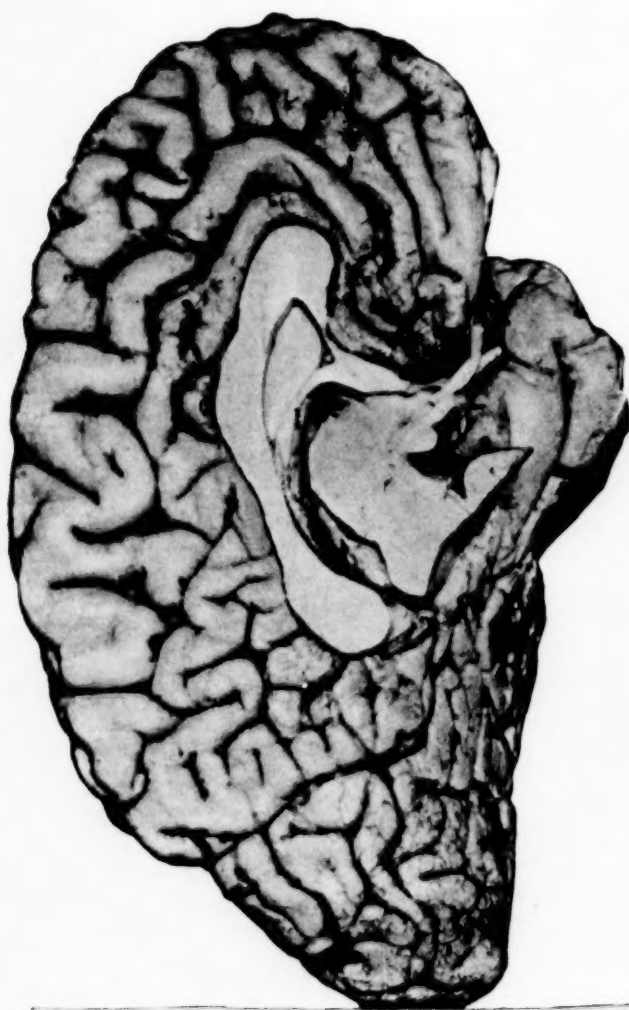




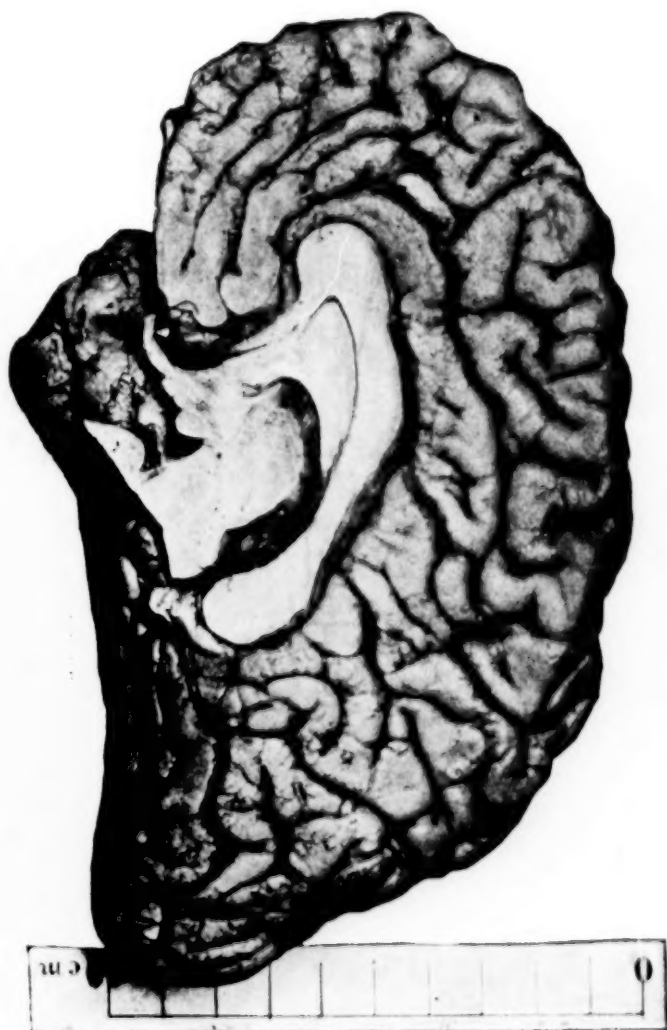


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## Notes and Comment.

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### BULLETIN OF THE MASSACHUSETTS STATE BOARD OF INSANITY.

—This newly organized board has begun to send out monthly bulletins of its operations, a form of publication which seems a great improvement upon the practice of similar boards, which have generally contented themselves with communicating with institutions quarterly or sometimes only annually. This more frequent publication keeps all officers of institutions and other interested persons promptly informed as to what is being accomplished throughout the state, and elsewhere.

In the bulletin for November we find the announcement that the trustees of a state hospital are being urged to consider the advisability and practicability of establishing a training school for nurses.

We are informed further that a committee of three experienced superintendents has been appointed to study the problem of standardizing the salaries of all officers and employes in institutions under the charge of the board, and to report the results of their studies.

Steps have been taken also to transfer inebriate women to a single hospital, so that all may be under treatment in one place. This would seem to promise more uniform methods of treatment and a reduction to a minimum of the discomfort incident to such patients, who are commonly a disturbing element if treated with insane patients.

Besides such administrative details, an interesting report is given of social-service work in the several hospitals. Some of the details, it must be confessed, seem unnecessarily minute, as, for example, an account of the disposition of a small kitten which had been found in an empty house by a social-service worker. All will be relieved to know that "a home was obtained and the little animal taken there; otherwise it would have been taken to the *Animal Rescue League*."

The statistics of the Psychopathic Hospital show that 115 patients had been admitted during the month of October. The sources of supply seem to have been varied; two came from schools, seven from courts, 33 from hospitals, 20 from charities, 36 were discharged house-cases reporting for the first time, seven were patients brought by their physicians, and 10 came on their own initiative.

In the matter of forms of mental disease thus admitted, it is interesting to notice that 12 were cases of alcoholism, or of alcoholism plus another psychosis; 16 were feeble-minded; the diagnosis in 22 cases was deferred, and in 19 cases no disease was found—a total of 69 out of 115 cases, or 60 per cent. It would appear from these statistics that feeble-minded and alcoholic cases promise to constitute a large percentage of the cases admitted to such a hospital.

Another interesting branch of work of increasing importance is the effort made by the superintendents of hospitals to establish clinics for the benefit of patients sent to them by physicians. Thus, for example, during the month of October two such clinics were held at Springfield and Pittsfield. That at Springfield was attended by the superintendent of the Munson State Hospital and a nurse, and by the superintendent and assistant superintendent of the Northampton State Hospital. At these clinics patients were seen and examined in the presence of their physicians, and advice was given as to their treatment. Three patients who were not present had their cases presented by their physicians, and 15 other patients received advice through personal consultation with their relatives.

A similar statement is made as to the work done by the officers of the Massachusetts School for the Feeble-Minded, where, upon Thursday of each week, it is customary for the superintendent and medical staff of the school to examine patients presented for the purpose. The number of such patients varies weekly from three to 12. In the month of October, 47 such cases were dealt with; 21 were examined, a diagnosis was made, treatment was indicated and a prognosis was given. Seven cases not present were described by letter. In six other cases presented by parents or guardians who made personal visits, advice was given. Thirteen former patients returned to report their progress. Of the 21 who

were examined, seven were brought by physicians, five by parents or guardians, four from public charities, three from private charities, one from the public schools and one from the Juvenile Court.

These various items indicate the scope of the bulletin and the amount of good which can come from thus informing institutions and medical officers of the state, and citizens generally, as to the facilities existing for the study of mental defect or the treatment of mental disease.

THE SHAME OF NEW HAMPSHIRE.—While Governor Whitman, of New York, is credited with having declared in a recent utterance, at once incisive and cheering, that "politics does not mix well with health and charity," the State of New Hampshire, through its Board of Control, has been illustrating the incompatibility of those ingredients in an attempt, for the moment unhappily successful, to oust from office one of the most efficient servants of the commonwealth.

Alas, thanks to a despicable intrigue, Dr. Charles P. Bancroft, for over thirty-two years Superintendent of the New Hampshire State Hospital at Concord, a distinguished alienist and publicist, a man of highest character, and a former President of the American Medico-Psychological Association, was deposed on December 31, 1914, after having declined to resign in default of specific charges. The details of this pot-house plot are incredibly sordid and constitute an anachronism in the management of public institutions in New England. The out-going executive, Governor Felker, who was a member ex-officio of the Board of Control, asked Mr. B. W. Couch, also a member, to request Dr. Bancroft to hand in his resignation. As the governor declined to assign any reason for this request, Mr. Couch refused to act. On the following day, December 7, the board, voting three to two, demanded Dr. Bancroft's resignation, to take effect on or before December 18. Those voting "Aye" were Governor Felker, George W. Fowler, purchasing agent, and Dr. George W. McGregor, of Littleton, N. H., these members having been vigorously opposed by Messrs. Couch and William J. Ahern. On December 8 Dr. Bancroft, by letter, requested the board to give its reasons, and as no satisfying reply was received his resignation was not forthcoming on that arbitrary demand. On December 18 the board met and voted as before,



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dismissal to occur, in default of resignation, on December 31. Between December 18 and 31 the board received two applications for the position, one of which was withdrawn when the candidate became aware of all the facts in the case, and the other, that of Dr. Edgar O. Crossman, of Lisbon, N. H., who at the recent state election had been chosen as senator. This name will be new to readers of this JOURNAL as that of a psychiatrist. We are informed that Dr. Crossman saw service at the Vermont State Hospital as an attendant, that he was on the staff of the Clifton Springs Sanatorium, N. Y., and four years Superintendent of the Markleton Sanatorium, Pa. He was at one time Collector of Internal Revenue at Portsmouth, N. H., and according to a Concord despatch to the *Boston Herald*, "He holds a prominent and enviable position in both medical and political circles in this state." We have said enough to suggest the quality of the aspirant's equipment as compared with that of Dr. Bancroft.

Of January 1 Mr. William J. Ahern, Secretary of the State Board of Charities and Corrections and a member of the Board of Control, and Mr. Couch filed a bill in equity which included allegations to the effect that Dr. Crossman was not qualified for the position. Accompanying this bill was a petition for a temporary injunction, on which the court ordered a hearing to be held on Saturday, January 9. On Monday, January 4, Dr. Crossman filed a letter of declination and at the meeting of the Board January 8 the following vote was passed: "Voted that the declination of Dr. E. O. Crossman to take the superintendency of the hospital be accepted and because of his voluntary declination the votes of December 31 electing him to that position, authorizing the secretary to make a contract with him, and fixing the time when his salary shall commence, are rescinded." It is understood that on January 9 the petitioners voluntarily withdrew their application for a temporary injunction, which withdrawal was followed by the request that the suit itself be dismissed. Meanwhile, Dr. Charles H. Dolloff, for a long time assistant superintendent, has been appointed acting superintendent.

It is a great satisfaction to the friends of the dependent insane of New Hampshire that the effort to protect the New Hampshire State Hospital from the installation of Dr. Crossman as superintendent during the last hours of the administration of Governor

Felker, has been successful. This was accomplished by obtaining the order for a hearing as late as January 9, which was two days after the new governor, the Hon. Rolland H. Spaulding, took office and became automatically a member of the Board of Control. The state legislature is in session and it is probable that a resolution will be introduced in the near future authorizing and instructing some committee to make an investigation of the circumstances and reasons for the summary dismissal of Dr. Bancroft. And if in the process of this investigation evidence should be disclosed on which facts could be found which put the efficiency of the management of the institution in question, thereupon such committee would investigate the question thus raised. Appeals have been made to Governor Spaulding, to reinstate Dr. Bancroft, by prominent men in and out of the medical profession throughout New England. As a sample of such appeals one may quote the following excerpt from a letter addressed by President Faunce, of Brown University, to the Secretary of the State Board of Control, under date December 17, as follows:

All competent physicians outside the State of New Hampshire are alarmed and dismayed by the proposed removal of Dr. Bancroft, and all the educational authorities of New England will be discouraged when they hear of it. Everyone knows that the intrusion of politics into the care of the insane is dangerous, not only to the inmates of the institution but to the entire community, and discouraging to all workers for the public good. It is the belief of thousands outside of the State of New Hampshire that aside from political influence Dr. Bancroft is universally respected and honored. I beg your honorable board to consider the possible result if he is driven from his post.

I trust my frank expression of opinion may not seem unwarranted, in view of my own childhood and my regard for the great work that has been done for the afflicted in the state hospital at Concord.

The people of New Hampshire have given vent to their righteous indignation in the press and by individual letters to the new governor in all parts of the state, and psychiatrists throughout the country are in arms against the chicanery of a wretched cabal that would deprive the State of New Hampshire, for no discoverable reason, of a capable and faithful public servant, whose work will live and whose name will be cherished in the hearts of his colleagues long after the pusillanimous persecution of his detractors shall have ceased to perturb the memories of just and decent men.



Meanwhile men may well ask themselves the question whether the old-fashioned, respectable, *unpaid* boards of management of an earlier day have not many points of superiority over new-fangled boards of control, a frequent disability of which is an incapacity to control themselves.

THE DEPENDENT INSANE IN RHODE ISLAND.—It is refreshing to turn from the disgrace of New Hampshire to what seems to be the promise of good things for Rhode Island. For a long time the state administration of the insane has been unpleasantly associated with that of the penal institutions and a Board of Supply and Control, of recent creation, has been attempting to make that connection still more intimate. The medical profession is agitating for the establishment of a board of insanity composed of unpaid members, with a salaried executive officer, which shall have exclusive control of the dependent insane and defectives. It is hoped that the new governor, the Hon. R. Livingston Beeckman, may favor such legislation. At all events, His Excellency's reference to the insane in the excerpt from his inaugural message, which follows, is encouraging in so far as it breathes an unmistakable sympathy with the cause for which physicians of Rhode Island are pleading:

The dependent insane and the mentally defective, as the wards of the state, make to the human heart an appeal that should always find expression in generous provision for their welfare. It is apparent that Rhode Island has not reached a standard of efficiency in caring for and treating these suffering people either adequate or satisfactory, or such as obtains in neighboring commonwealths. It is proper that we should consider where lies the path of duty and progress. Before all else, it is important, as a measure of mere decency, that the chain that now binds the affairs of the reformatory and penal institutions with those of the state hospital for the insane should be severed. By the vicious union that now exists, we stigmatize mental disease—the greatest of human hardships—as connected with crime.

The medical aspects of hospital treatment should be kept constantly in view and nothing be left undone that might promote recovery and bring cheer into the lives of a multitude of men and women at Howard, who, in nearly all cases through no fault of their own, have fallen by the wayside in the struggle for existence, and whose plight calls for aid as eloquently as that of any soldier who has been left wounded on the field of battle.



THE OCCUPATIONAL DIVERSION EXHIBIT.—The committee in charge of the exhibit to be held at the seventy-first annual meeting of the American Medico-Psychological Association, Hotel Chamberlain, Old Point Comfort, Va., May 11-14, 1915, has already formulated plans for it and sent out letters asking the cooperation of hospitals in making it a success. The three exhibits which have been held have sufficiently shown the value of gathering together specimens of various forms of handicraft. The attendance at these indicated that all are interested in this important form of treatment and are anxious to gain ideas from the experience of others. It is hoped that the new departure of grouping specimens of the same class of work together, and not as an exhibit of a particular hospital, will do much to increase the number of exhibitors. Under the new plan a single piece will be as conspicuous as will several. Probably the hall will not be quite so attractive without the booths which have been a feature in the past, but it is hoped that the committee will be able to make up for this in other ways.

We learn that the committee has asked that there be time allotted on the program for a discussion of certain questions on the subject of occupational diversion. This will undoubtedly prove interesting and helpful to all.

It is hoped that every hospital in our membership will be represented by material showing at least one form of occupation. The groups for which certificates of merit will be given should bring forth some very interesting and helpful ideas.

## Book Reviews.

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*The Myth of the Birth of the Hero (A Psychological Interpretation of Mythology)*. Nervous and Mental Disease Monograph Series, No. 18. By DR. OTTO RANK of Vienna. Authorized translation by DRS. F. ROBBINS and SMITH ELY JELLIFFE. (New York: The Journal of Nervous and Mental Disease Publishing Company, 1914.)

One of the problems of mythical research "consists in the elucidation of the reason for the extensive analogies in the fundamental outlines of mythical tales, which are rendered still more enigmatical by the unanimity in certain details and their reappearance in most of the mythical groupings." The first theory to explain this is the "Idea of the People," propounded by Bastian, which assumes the existence of elementary thoughts, so that the unanimity of the myths is a necessary sequence of the uniform disposition of the human mind. The second is that of original community, and the third, or modern theory, that of migration or borrowing. An introduction discusses these theories and emphasizes the intimate relation between dream and myth. "King Œdipus, who has murdered his father Laios, and married his mother Iokaste, is merely the wish fulfilment of our childhood." The author believes that this relation is confirmed by the narration of the myths which follow. These are of Sargon, Moses, Karna, Œdipus, Paris, Telephos, Perseus, Gilgames, Kyros, Tristan, Romulus, Hercules, Jesus, Siegfried, and Lohengrin. From these a standard saga may be formulated as follows: The hero is the child of most distinguished parents; usually the son of a king. His origin is preceded by difficulties, such as continence, or prolonged barrenness, or secret intercourse of the parents, due to external prohibition or obstacles. During the pregnancy, or antedating the same, there is a prophecy, in form of a dream or oracle, cautioning against his birth, and usually threatening danger to the father or his representative. As a rule, he is surrendered to the water, in a box. He is then saved by animals, or by lowly people (shepherds), and is suckled by a female animal, or by a humble woman. After he has grown up, he finds his distinguished parents, in a highly versatile fashion; takes his revenge on his father, on the one hand, is acknowledged on the other, and finally achieves rank and honors.

The normal relations of the hero towards his father and his mother regularly appear impaired in these myths. Certain difficulties appear when we search for the motive for this, due to the fact that we do not fully understand the imaginative faculty of the child. Investigation of this

faculty has hardly commenced. The psychoanalytic method which has been developed through the work of Freud is the only known instrument which makes this study practicable.

The book is a most interesting one and is well worth perusal, whether one agrees with the theories and conclusions or not. An excellent index adds to its value.

W. R. D.

*Mental Medicine and Nursing.* For Use in Training-Schools for Nurses and in Medical Classes and a Ready Reference for General Practitioners. By ROBERT HOWLAND CHASE, A. M., M. D., Physician-in-Chief Friends Asylum for the Insane, Frankford, Philadelphia, etc. (Philadelphia and London: J. B. Lippincott Company.)

This book is divided into five parts, has an appendix and is prefaced with a brief glossary of terms. Part one is devoted to a brief account of the anatomy of the nervous system, with very little reference, however, to its physiology, an unfortunate omission in a work of this character. If any account of the structure of the nervous system was considered necessary surely some reference to the accepted views of function should have been included. Following the sixteen pages dealing with the nervous system are thirty-five which are devoted to what may be termed physiological-psychology and which forms the second part of the book. In this part some of the physiology of the nervous system, the omission of which is noted in the first part, is considered along with a discussion of mental processes, complexes, dreams, autistic thinking, and the like. Then follows part three, dealing with insanity, general symptomatology, disturbances of mental elaboration, of feelings and emotions, and of volition and action. Part four treats of the forms of mental disorder and part five with the patient from the physician's view-point, the patient from the nurse's view-point, food, feeding, etc. The appendix considers poisons and the treatment of poisoning.

It is to be regretted that the author did not retain his original purpose of preparing a text-book upon mental disorders "that could be used conveniently by pupil-nurses attending lectures on this subject." We feel that in extending "the scope of the work to meet the needs of the busy practitioner and other students" he has missed, in some degree, meeting either the requirements of nurses or of the busy practitioner. Many things concerning which the busy practitioner would like information are necessarily omitted, and many points concerning which nurses should be informed are lacking.

One is surprised to find paresis included among auto-intoxication psychoses, and the general consideration of the symptomatology, etiology and treatment of the disease leaves much to be desired. The author apparently has not permitted himself to accept syphilis as the primary cause of the disease. Nothing is said of lumbar puncture now a routine method in making an examination of a suspected case, nor is reference made to the observations of Moore and Noguchi or the method of treat-

ment of Swift and Ellis. The account of dementia præcox, which is also grouped with auto-toxic psychoses, is too brief to be of value to students or practitioners seeking information and too condensed to be grasped by nurses.

The work is rather profusely illustrated but the application of some of the illustrations may be called in question. For example, a picture on page 78 of a man with a luxuriant beard is given as a "common type of systematized delusions." One on page 76 is presented to illustrate "flight of ideas; the rambling form." When the picture was taken the patient was a chronic dement with too few ideas to permit a flight. On page 58 is a picture taken from Berkley's Treatise on Mental Diseases under which is the legend, "A common type of climacteric psychosis." In Berkley's book this picture is one of two of the same patient said by the author to be a case of periodic mania; the picture which has been copied showing the patient, according to Dr. Berkley, "after she had attained to a degree of mild dementia."

In the selection of pictures from other publications Dr. Chase has been, with a few exceptions, careful to give credit to the original source, but it is to be regretted that a more careful use of quotation marks and a more frequent reference to the source of some of the material which has been incorporated in the book was not made. The remarks on the photography complex on page 47 are taken, in part, *verbatim* from page 61 of Hart's Psychology of Insanity recently reviewed in this JOURNAL.

On page 199 the prospectus of a course of lessons "to qualify nurses to amuse patients or give them simple occupation and to train nurses in methods suitable for the re-education of patients" is not the prospectus as stated in the book of a school of civics or philanthropy, but of the occupation course in the nurses' training school of a well-known hospital for nervous and mental diseases. So also on page 201 the example of a regular schedule in the foot-note is taken from the twentieth annual report (1911) of this same hospital, with the single change of the case number from 2317 to 4545 with no credit whatever to the place of original publication.

The statement of the relative merits of nasal and œsophageal feeding on page 214, although arranged in the work under review in a different manner, will be found stated in almost identical language and in the same sequence on pages 439-440 of Craig's Psychological Medicine. The glossary of terms which prefaces the book will be found of use but needs some correction. For instance, we should not call *délire à deux* "the morbid influence of one disordered mind on that of another producing insanity," but rather the condition produced. *Hæmatoporphyrinuria* is not "blood in urine" and it is due to other things than the "unwise use of sulphonal." The definitions of neologism and verbigeration are the same: "the repetition of senseless expressions"—surely the author in thus defining neologism has confused the thing expressed with its expression.

The book on the whole is well written and its defects are due largely

to an attempt to compress a great deal in a small space. It will have its uses in training schools as a book for reading to supplement lectures and bedside instruction.

*Serology of Nervous and Mental Diseases.* By D. M. KAPLAN, M. D.,  
Director of Clinical and Research Laboratories of the Neurology  
Institute, New York City. (Philadelphia and London: W. B. Saunders  
Company, 1914.)

This timely work by Dr. Kaplan is a volume of some two hundred and sixty-three pages of text, to which are added seventy-one pages giving the literature of the subject and an index of twelve pages.

The book is divided into four parts. Part one, Technology, gives a brief statement of the history of lumbar puncture, an account of the anatomy and physiology of the structures involved in the procedure and then the indications and contraindications for the operation, the preparation of the patient, the technic of lumbar puncture, the phenomena attending it, the after care of the patient and the disposal of the fluid obtained. Then follow several pages devoted to general considerations of the spinal fluid, its physical properties, chemical characteristics, cytology, with methods of cell counting and interpretation of results. Serology is next considered, with an account of the modifications of the Wassermann reaction, controls and their significance, and the performance of the Wassermann reaction.

Part two treats of the serology of nervous and mental diseases of non-luetic etiology. Part three of the serology of nervous and mental diseases of luetic origin, and part four of the therapeutic use of salvarsan. The author clearly believes in approaching every case with an open mind, and therefore says that "in submitting a serum for analysis it is important, in order to obtain an unbiased opinion from the laboratory, to withhold the clinical findings in the case." He advises all who wish to engage in experimental work to reject all information that might interfere with an unbiased and reliable conclusion.

Throughout the work the directions are most carefully given and the author does not hesitate to withhold judgment as to certain findings or deductions of others where those do not correspond with his own observations or do not appear to be based upon sufficient or satisfactory observation. For example, he does not appear to regard the colloidal gold reaction of as general application in distinguishing different forms of cerebral lues as do some observers.

The work is of such a character that only a more extended review than space or time permits will do it justice. It will, we predict, find a ready welcome in clinical laboratories and among clinicians as well as laboratory workers. The work shows painstaking care in its preparation, is clear and precise in its directions and bears the stamp of a conscientious observer. The preparation of the book in clearness of type and illustration reflects credit upon the publisher. But few proof errors have met our notice. Korsakoff on page 124 is Korsakow on page 110. Clue is evidently the word meant by "cue" on page 130.